WEST Search History

DATE: Friday, November [illegible]

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L14	L2 and L12	2	L14
L13	L1 and L12	0	L13
L12	GGGACTTTCC	64	L12
L11	L2 and L9	184	L11
L10	L1 and L9	101	L10
L9	binding sites	37632	L9
L8	L6 and L2	0	L8
L7	L6 and L1	1	L7
L6	L3 and L5	64	L6
L5	dendritic cell	3007	L5
L4	ribozym$3	8755	L4
L3	tolerogen$3	395	L3
L2	nf kappa b	424	L2
L1	nuclear factor kappa b	211	L1

END OF SEARCH HISTORY

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      (FILE 'HOME' ENTERED AT 11:34:48 ON 21 NOV 2002)

      FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 11:34:[illegible] ON 21 NOV 2002
L1         25547 NUCLEAR FACTOR KAPPA B
L2         41469 NF KAPPA B
L3          4421 TOLEROGEN?
L4         15153 RIBOZYM?
L5        183195 OLIGONUCLEOTIDE
L6         77082 ANTISENS?
L7           621 L3 AND L4
L8             1 L7 AND L5
L9             1 L7 AND L6
L10            1 L8 OR L9
L11                [illegible]
L12            1 L10 [illegible] AND L1
L13            1 L12 [illegible]
L14            1 L13 AND L2 AND L5
L15            1 L14 AND L6
L16          382 [illegible] SEL
L17        44186 [illegible] SEL
L18            1 L1 AND L2 AND L17
L19            1 DUP REM L16 (1 DUPLICATE REMOVED)
L20            1 L2 AND L5 AND L17
L21           15 DUP REM L20 (6 DUPLICATES REMOVED)

      FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 11:4[illegible] ON 21 NOV 2002
L22            7 L17 AND L3 AND L1
L23            5 DUP REM L22 (2 DUPLICATES REMOVED)
L24           10 L17 AND L1 AND L6
L25            6 DUP REM L24 (4 DUPLICATES REMOVED)
L26            [illegible] L17 AND L1 AND L5
L27                L26 NOT L25

      FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 11:4[illegible] ON 21 NOV 2002

      FILE 'REGISTRY' ENTERED AT 11:[illegible] ON 21 NOV 2002
L28           [illegible]

      FILE 'CAPLUS' ENTERED AT 11:[illegible] ON 21 NOV 2002
L29                [illegible] L28 AND L17
L30           914 RIBOZYME/BI
L31             [illegible] L1 AND L2
L32             814 L2 AND L30
L33               6 L31 AND L17
L34               6 DUP REM L33 (0 DUPLICATES REMOVED)
L35               8 L32 AND L17
L36               8 DUP REM L35 (0 DUPLICATES REMOVED)

=>
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L34 ANSWER 1 OF 4 [illegible] COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible] [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Selective expression of type I IFN genes in human **dendritic cells** infected with Mycobacterium tuberculosis
AUTHOR(S): Remoli, Maria Elena; Giacomini, Elena; Lutfalla, Georges; Dondi, Elisabetta; Orefici, Graziella; Battistini, Angela; Uzé, Gilles; Pellegrini, Sandra; Sibirin, Filiana M.
CORPORATE SOURCE: Laboratories of Immunology, Istituto Superi[illegible] di Sanita, Rome, I-[illegible], Italy
SOURCE: Journal of Immunology ([illegible]), [illegible], 3[illegible]-[illegible]
[illegible]; [illegible]
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Type I IFN [illegible] different aspects of the immune response, inducing a cell-mediated immunity. We have recently shown that the infection of **dendritic cells** [illegible] with Mycobacterium tuberculosis (Mtb) induces IFN-.alpha.. In this work we have monitored a rapid induction of IFN-.beta. followed by the delayed prodn. of the IFN-.alpha.1 and/or -.alpha.13 subtypes. The Mtb infection rapidly activates the NF-.kappa.B complex and stimulates the phosphorylation of IFN regulatory factor (IRF)-3, events known to induce IFN-.beta. expression in viral infection. In turn, the autocrine prodn. of IFN-.beta. induces the IFN-stimulated genes that contain **binding sites** for activated STATs in their promoters. Among the IFN-stimulated genes induced in DC through STAT activation are IRF-1 and IRF-7. The expression of IRF-1 appears to be dependent on the sequential activation of NF-.kappa.B and STAT-1. Once expressed, IRF-1 may further stimulate the transcription of IFN-.beta.. Induction of IRF-7 is also regulated at the transcriptional level through the binding of phosphorylated STAT-1 and STAT-2, forming the IFN-stimulated gene factor 3 complex. In turn, the IRF-1 and IRF-7 [illegible] are required for the amplification of the autocrine IFN-.alpha./.beta. [illegible]. Although preliminary, our results strongly suggest that the selective induction of IFN-.alpha. genes in Mtb-infected DC [illegible] primarily expressing NF-.kappa.B and IRF-3 are activated and contribute to the rapid IFN-.beta. expression. In turn, IFN-.alpha./.beta. and IRF-1 and IRF-7 may cooperate toward induction of IFN-.alpha. genes [illegible] infection persists and these factors are activated.
REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER [illegible] OF 4 [illegible] COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible] [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: The use of [illegible] **dendritic cells** for enhancing tolerogenicity in a host and methods for making the same
INVENTOR(S): Robbins, Paul D.; [illegible], Lina; [illegible], Hong
PATENT ASSIGNEE(S): [illegible] of [illegible] [illegible] System, Minneapolis, MN
SOURCE: [illegible], [illegible]
[illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: English
FAMILY [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2001-844915 20010427

PRIORITY APPLN. INFO.: US 2000-200479P P 20000428

AB The present invention relates to a tolerogenic mammalian **dendritic cells** (DCs) and methods for the prodn. of the tolerogenic DCs. In addn., the present invention provides a method for enhancing tolerogenicity in a host comprising administering the tolerogenic mammalian DCs of the present invention to the host. The tolerogenic DCs of the present invention comprise an oligodeoxyribonucleotide (ODN) which has one or more NF-.kappa.B **binding sites**. The tolerogenic DCs of the present invention may further comprise a viral vector, and preferably an adenoviral vector, which does not affect the tolerogenicity of the tolerogenic DCs when present therein. Enhanced tolerogenicity in a host is useful in prolonging foreign graft survival and for treating inflammatory related diseases, such as autoimmune diseases.

L34 ANSWER 2 OF [illegible] ON STN

ACCESS ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] cardiac allograft survival using **dendritic cells** treated with NF-.kappa.B decoy oligodeoxyribonucleotides

AUTHOR(S): Giannoukakis, Nick; Bonham, C. Andrew; Qian, Shiguang; Chu, Zhongyou; Peng, Lansha; Harnaha, Jo; Li, Wei; Thomson, Angus W.; Fung, John J.; Robbins, Paul D.; Ni, Lina

CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA

SOURCE: Molecular Therapy (2000), 1(5, Pt. 1), 430-437
CODEN: MTOHCK; ISSN: 1525-0016

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB **Dendritic cells** [illegible] response [illegible] type [illegible] regulation [illegible] cells. [illegible] cells, [illegible] DCs, [illegible] binding activity to [illegible] is assocd. with [illegible] proteins. [illegible] NF-.kappa.B-dependent [illegible] translocation [illegible] that double-stranded oligodeoxynucleotides contg. NF-.kappa.B **binding sites** for NF-.kappa.B, NF-.kappa.B p50 are efficiently incorporated by bone marrow-derived DCs and significantly inhibit NF-.kappa.B-dependent transcription of a reporter gene. Moreover, exposure of DCs to the oligodeoxynucleotide decoys inhibits lipopolysaccharide (LPS)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B [illegible] cell-surface expression of costimulatory molecules or interleukin-12 MHC class I or class II expression. Furthermore, NF-.kappa.B [illegible] induced allogeneic T cell-proliferative responses [illegible] Finally, [illegible] NF-.kappa.B [illegible]

significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways involved in immune stimulation in DC using DN decoy approaches could be one means to promote tolerance induction in organ transplantation. (c) 2000 Academic Press.

REFERENCE COUNT: 74 THERE ARE 74 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 1 OF [illegible]
ACCESS NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] dendritic cell [illegible] through the inhibition of nuclear factor-. kappa.B activation in hemopoietic progenitor cells

AUTHOR(S): [illegible]yama, Tsutomu; Han, Sophia; Ishida, Tadao; Nadaf, Sirena; Kerr, Lawrence; Carbone, David P.; Gabrilovich, Dmitry I.

CORPORATE SOURCE: The Vanderbilt Cancer Center and Departments of Medicine and Microbiology and Immunology, Vanderbilt University School of Medicine, Nashville, TN, 37232, USA

SOURCE: Journal of Immunology (1998), 160(3), 1224-1232
CODEN: JOIMA3; ISSN: 0022-1767

PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB [illegible] dendritic cells [illegible] binding sites for VEGF decreased [illegible] nuclear factor-. kappa.B NF-.kappa.B-dependent activation of reporter gene transcription during the first 24 h in culture. The presence of VEGF significantly decreased the specific DNA binding of NF-.kappa.B as early as 30 min after incubation with TNF-.alpha.. This was followed on days 2 to 10 by decreases in the mRNA for RelB and c-Rel, two subunits of NF-.kappa.B. Blockade of NF-.kappa.B activity in HPC at early stages of differentiation with an adenovirus expressing a dominant I.kappa.B inhibitor of NF-.kappa.B reproduced the pattern of effects induced with VEGF. Thus, NF-.kappa.B plays an important role in maturation of HPC to DC, and VEGF activity at the Flt-1 receptor is likely to result in activation of NF-.kappa.B in this system. [illegible] of NF-.kappa.B activation in HPC by tumor-derived factors may [illegible]

L44 ANSWER [illegible]
ACCESS NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] primary cultures of NF-.kappa.B subunits for Ik-Ik [illegible]

AUTHOR(S): [illegible]; Bellavia, Maria L.; Bianchi, [illegible]; [illegible]; Ayt Lh, Emita; Fiorucci, Maria L.; [illegible], Ivo

CORPORATE SOURCE: [illegible] Biologia, [illegible], Rome, Italy

SOURCE: Immunity [illegible], [illegible]

[illegible]; [illegible]

[illegible]:
[illegible]:
[illegible]:
AB [illegible] IL-12 receptor by fresh **dendritic cells** [illegible]. Using RT-PCR, RNAse protection, and electrophoretic mobility shift assay anal., they found that DC possess an IL-12 receptor with .beta.1 subunit (downstream box 1)-related differences from that on T cells. IL-12 signaling through this receptor involved members of the NF-.kappa.B but not STAT family. The unique properties of the IL-12 receptor in DC, characterized by a single class of **binding sites** with a Kd of about 300 pM, may underlie rather unique effects, such as IFN.gamma.-independent augmentation of class II antigen expression and priming for IFN-induced prodn. of IL-12.
REFERENCE COUNT: 48 THERE ARE 48 [illegible] AVAILABLE FOR THIS RECORD. ALL [illegible]

134 [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] transcript by CD40 [illegible] **nuclear factor-.kappa.B**
AUTHOR(S): [illegible]; Nagase, Hisashi; Ishida, Takaomi; [illegible]; Mukaida, Hideo
CORPORATE SOURCE: Department Allergology, Institute Medical Science, University Tokyo, Tokyo, 108, Japan
SOURCE: European Journal of Immunology (1997), 27(12), 3461-3471
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Interleukin-12 is produced in response to infection with bacteria or parasites or to bacterial cell-wall components such as lipopolysaccharide (LPS) in monocytes/macrophages and **dendritic cells**, [illegible] generated by the interaction between [illegible] antigen-presenting cells [illegible]. Transcriptional analysis [illegible] LPS [illegible] such as [illegible]. Transcription was [illegible] by CD40 ligation was characterized in [illegible], Raji, and a human [illegible] cell line, WIL-2. These cells, stimulated by an [illegible] by transfection with a CD40L [illegible], [illegible] enhanced p40 mRNA expression. [illegible] potential nuclear [illegible] NF-.kappa.B **binding sites** [illegible]. Gel mobility shift assay revealed that the [illegible] NF-.kappa.B complex which is located around 120 bp upstream of the transcription initiation site in murine and human p40 genes formed an NF-.kappa.B complex with nuclear ext. from Daudi cells stimulated by CD40 ligation. Moreover, transfection of Daudi cells with the polymd. NF-.kappa.B binding sequence linked to a thymidine kinase/chloramphenicol acetyltransferase (CAT) expression plasmid greatly induced CAT activity, but transfection with the polymd. mutated NF-.kappa.B binding sequence did not. These results indicate that the NF-.kappa.B [illegible] is [illegible] and [illegible] to NF-.kappa.B.

L36 ANSWER 1 OF 7 [illegible]

ACCESSION NUMBER: 2002:712316 CAPLUS

TITLE: Marked prolongation of cardiac allograft survival by **dendritic cells** genetically engineered with NF-.kappa.B oligonucleotide [illegible]

AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible]

SOURCE: Journal of Immunology (2002), 169(6), 3382-3391

CODEN: JOIMA3; ISSN: 0022-1767

PUBLISHER: American Association of Immunologists

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Bone marrow-derived **dendritic cells** (DCs) can be genetically engineered using adenoviral (Ad) vectors to express immunosuppressive mols. that promote T cell unresponsiveness. The success of these DCs for therapy of allograft rejection has been limited in part by the potential of the adenovirus to promote DC maturation and the inherent ability of the DC to undergo maturation following in vivo administration. DC maturation occurs via NF-.kappa.B-dependent mechanisms, which can be blocked by double-stranded "decoy" oligodeoxyribonucleotides containing NF-.kappa.B binding sites (i.e. NF-.kappa.B. [illegible] NF-.kappa.B [illegible] Ad-CTLA4-Ig [illegible] DCs exhibit markedly impaired allostimulatory activity and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL10; B1 (H-2b)) before transplant significantly prolongs MHC-mismatched (BALB/c; CBA (H-2d)) vascularized heart allograft survival, with long-term (>100 days) donor-specific graft survival in 4/5 of recipients. The mechanisms responsible for DC tolerogenicity, which may involve activation-induced apoptosis of alloreactive T cells, do not lead to generalized immunosuppression in vivo. Taken together, NF-.kappa.B antisense decoys in conjunction with Ad encoding a potent costimulation blocking agent offer a promising new therapy of allograft rejection or autoimmune disease with minimal risk of systemic immunosuppression.

REFERENCE COUNT: [illegible] THERE ARE [illegible] RECORDED REFERENCES AVAILABLE FOR THIS RE. ALL REFERENCES AVAILABLE IN THE RE FORMAT

L36 ANSWER [illegible]

ACCESSION NUMBER: [illegible]

TITLE: [illegible] dendritic cells [illegible]

AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible]

SOURCE: [illegible]

PUBLISHER: [illegible]

L[illegible] TITL:
LANGUA :
AB [illegible] **dendritic cells** [illegible] NF-.kappa.B [illegible] binding sites [illegible] IRF-1 and IRF-7. [illegible] NF-.kappa.B [illegible] STAT-1. [illegible] IFN-.beta.. [illegible] IFN-.alpha.1/13 [illegible]

REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L36 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:810872 CAPLUS
DOCUMENT NUMBER: 135:355016
TITLE: The use of tolerogenic **dendritic cells** for enhancing tolerogenicity in a host and methods for making the same
INVENTOR(S): Robbins, Paul D.; [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
L[illegible] TYPE:
LANGUA :
FAMILY [illegible]
PATENT [illegible]

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]			[illegible]	
[illegible]				

DESIGNATED STATES: [illegible]

AB [illegible] **dendritic cells**

... tolerogenicity in ... mammalian DCs of the present invention ... of the present invention comprise an oligodeoxyribonucleotide (ODN) which has one or more **NF-.kappa.B binding sites.** The tolerogenic DCs of the present invention may further comprise a viral vector, and preferably an adenoviral vector, which does not affect the tolerogenicity of the DCs of the present invention. ...

...

ACCESSION NUMBER: ...

DOCUMENT NUMBER: ...

TITLE: ... dendritic cells ... NF-.kappa.B ... oligodeoxyribonucleotides

AUTHOR(S): Giannoukakis, Nick; Bonham, C. Andrew; Qian, Shiguang; Zhou, Zhongyou; Feng, Lansha; Harnaha, Jo; Li, Wei; Thomson, Angus W.; Fung, John J.; Robbins, Paul D.; Lu, Lina

CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA

SOURCE: Molecular Therapy (2000), 1(5, Pt. 1), 430-437 CODEN: MTOHCK; ISSN: 1525-0016

PUBLISHER: Academic Press

DOCUMENT TYPE: ...

LANGUAGE: ...

AB Dendritic cells ... NF-.kappa.B- ... NF-.kappa.B- ... NF-.kappa.B ... binding sites for NF-.kappa.B (NF-.kappa.B ODN) are efficiently incorporated by bone marrow-derived DC and specifically inhibit NF-.kappa.B-dependent transcription of a reporter gene. ... oligonucleotide decoys inhibited ... DC progenitors with NF-.kappa.B ... NF-.kappa.B ... NF-.kappa.B ... NF-.kappa.B ...

REFERENCE COUNT: ... THERE ARE ... REFERENCES AVAILABLE FOR THIS ...

L[illegible] ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] multiple TRAFs in the signaling [illegible] activator of NF-kappa.B, [illegible] of the TNFR superfamily

AUTHOR(S): Wong, Lawrence; Tometsko, Mark E.; Anderson, Dirk M.; Derose, David; Dougall, William C.
CORPORATE SOURCE: Department of Molecular Biology, Immunex Corporation, Seattle, WA, 98101, USA

SOURCE: Journal of Biological Chemistry (1998), 273(51), 34128-34133
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Receptor activator of NF-kappa.B (RANK) is a [illegible] member of the TNFR superfamily [illegible] dendritic cells. [illegible] RANK plays significant roles in the [illegible] dendritic cell [illegible] and differentiation. [illegible] demonstrate here the interaction of RANK with tumor necrosis factor receptor-associated factors (TRAFs) 1, 2, 3, 5, and 6 both in vitro and in vivo. Mapping of the structural requirements for TRAF/RANK interaction revealed multiple TRAF binding sites clustered in two distinct domains in the RANK cytoplasmic tail. These TRAF binding domains were shown to be functionally important for the RANK-dependent activation of NF-kappa.B and c-Jun NH2-terminal kinase activities. Site-directed mutagenesis also indicated that these TRAF binding sites exhibited selective binding for different TRAF proteins. In particular, TRAF6 interacted with membrane-proximal determinants distinct from those binding TRAFs 1, 2, 3, and 5. When this membrane-proximal TRAF6 interaction domain was deleted, RANK-mediated NF-kappa.B signaling was completely inhibited while c-Jun NH2-terminal kinase activation was partially inhibited. An NH2-terminal truncated mutant of TRAF6 inhibited RANK-mediated NF-kappa.B [illegible]

REFERENCE COUNT: [illegible] THERE ARE [illegible] REFERENCE(S) AVAILABLE FOR THIS [illegible] ARE NOT AVAILABLE IN THE FE FORMAT

L[illegible] ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] dendritic cell maturation through [illegible] activation

AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]

SOURCE: [illegible]

LANGUAGE:

AB Vascular endothelial growth factor (VEGF), produced by almost all tumor cells, affects the ability of bone marrow progenitor cells to differentiate into functional **dendritic cells** (DC) during the early stages of their maturation. In this study we demonstrate specific binding of VEGF to HPC. This finding was explained by placenta growth factor (PlGF), a VEGF-related protein that is also a Flt-1 receptor. The **binding sites** for VEGF were found during DC maturation in vitro and in vivo with increased levels of mRNA for Flt-1. VEGF also activated the transcription factor **NF-.kappa.B**. **NF-.kappa.B** was found to be constitutively active in HPC. In contrast, VEGF stabilized the inactive form of **NF-.kappa.B**. Activation of **NF-.kappa.B** was also inhibited by metalloproteases in the mRNA for both Flt-1 and **NF-.kappa.B**. Activation of **NF-.kappa.B** in HPC at early stages of differentiation with an adenovirus expressing a dominant I.kappa.B inhibitor of **NF-.kappa.B** reproduced the pattern of effects obsd. with VEGF. Thus, **NF-.kappa.B** plays an important role in maturation of HPCs to DC, and VEGF activation of the Flt-1 receptor is able to block the activation of **NF-.kappa.B** in this system. Blockade of **NF-.kappa.B** activation in HPCs by tumor-derived factors may therefore be a mechanism by which tumor cells can directly down-modulate the ability of the immune system to generate effective antitumor immune responses.

L36 ANSWER 7 OF 8 MEDLINE COPYRIGHT 2004 ACS
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE:
NF-.kappa.B
AUTHOR(S):
CORPORATE SOURCE:
SOURCE:
PUBLISHER:
DOCUMENT TYPE:
LANGUAGE:

AB The authors analyzed the expression of an IL-12 receptor by fresh **dendritic cells** (DC) and a DC line. Using RT-PCR, RNase protection, and electrophoretic mobility shift assay (EMSA), they found that DC possess an IL-12 receptor with structural features similar to IL-12 receptor-related differences from that on T cells. IL-12 stimulation induced receptor expression and activation of the **NF-.kappa.B** and not STAT factors. The ability of IL-12 to induce **binding sites**

REFERENCE COUNT:

L36 ANSWER 8 OF 8 MEDLINE COPYRIGHT 2004 ACS
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE:
NF-.kappa.B
AUTHOR(S):

[illegible]

[illegible]

[illegible]

PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]

AB Interleukin-12 (p40) is produced in response to interaction with bacteria or parasites or their bacterial constituents such as lipopolysaccharides (LPS) in monocytes/macrophages and **dendritic cells**, and also generated by the interaction between activated T cells and antigen-presenting cells via CD40-CD40 ligand (CD40L). Transcriptional analyses of p40 were carried out only using bacterial constituents such as LPS as stimuli. The transcriptional induction of p40 by CD40 ligation was characterized in a human B lymphoblastoid cell line, Daudi, and a human acute monocytic leukemia cell line, THP-1. These cells, stimulated by an agonistic monoclonal antibody against CD40, or by transfection with a CD40 expression vector, produced p40 mRNA and secreted p40 protein. Sequence analysis of the p40 promoter revealed a potential nuclear factor **NF-.kappa.B binding sites** [illegible] **NF-.kappa.B** binding sequence [illegible] bp upstream of the transcription initiation site in murine and human p40 genes formed an **NF-.kappa.B**-[illegible] from Daudi cells stimulated by CD40 ligation. Moreover, transfection of Daudi cells with the plasmid **NF-.kappa.B** binding sequence linked to thymidine kinase/chloramphenicol acetyltransferase (CAT) reporter plasmid greatly induced CAT activity, but transfection with the polymd. mutated **NF-.kappa.B** binding sequence did not. These results suggest that the **NF-.kappa.B** binding site located around 100 bp upstream of the transcription initiation site in murine and human p40 promoter regions could be important for the p40 induction by CD40 ligation via activation of **NF-.kappa.B**.

FILE 'HOME' ENTERED AT [illegible]

FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT [illegible]

L1 25547 NUCLEAR FACTOR KAPPA B
L2 4421 TOLEROGEN?
L3 44966 DENDRITIC CELL
L4 15153 RIBOTYM?
L5 13 L1 AND L4
L6 1 DUP REM L5 [illegible]
L7 [illegible]
L8 [illegible]
L9 [illegible]
L10 [illegible]
L11 [illegible]

FILE 'REGISTRY' [illegible]

L13 [illegible]

FILE 'CAPLUS' ENTERED AT 15:17:42 ON 21 NOV 2002

L14 16 L13 AND L1
L15 2 L14 AND L2
L16 16 DUP REM L14 (0 DUPLICATES REMOVED)

FILE 'BIOSIS, [illegible]' ENTERED AT 11:34:[illegible] ON 21 NOV 2002

L1 [illegible] L[illegible] AND [illegible]
L2 [illegible] L[illegible] AND [illegible]
L3 [illegible] L[illegible] AND [illegible]
L4 [illegible] L[illegible] AND [illegible]
L5 [illegible] OLIGONUCLEOTIDE
L6 77082 ANTISENS?
L7 622 L1 AND L6
L8 1 L7 AND L3
L9 1054 L2 AND L6
L10 3 L9 AND L4
L11 [illegible] DUP REM L[illegible] ([illegible] DUPLICATES REMOVED)
L12 [illegible] L[illegible] AND L[illegible] AND L[illegible]
L13 [illegible] L[illegible] AND L[illegible]
L14 [illegible] L[illegible] AND L[illegible]
L15 [illegible] L[illegible] AND L[illegible]
L16 [illegible] L[illegible] AND L[illegible]
L17 [illegible] [illegible] L[illegible]
L18 [illegible] L[illegible] AND L[illegible] AND L[illegible]
L19 [illegible] DUP REM L[illegible] L[illegible] ([illegible] DUPLICATES REMOVED)
L20 [illegible] L[illegible] AND L[illegible] AND L17
L21 [illegible] DUP REM L[illegible] ([illegible] DUPLICATES REMOVED)

FILE 'BIOSIS, MEDLINE, CAPLUS, EMBASE' ENTERED AT 11:49:58 ON 21 NOV 2002

L22 [illegible] L[illegible] AND L6 AND L1
L23 [illegible] DUP REM L[illegible] ([illegible] DUPLICATES REMOVED)
L24 10 L17 AND L3 AND L2
L25 6 DUP REM L24 (4 DUPLICATES REMOVED)
L26 0 L16 AND L3 AND L1
L27 0 L16 AND L3 AND L2

=>

119 ANSWER [illegible]

ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Involvement of MAPK, p38 and NF-kappaB signal transduction in regulation of TLR2, TLR4 and TLR9 gene expression induced by lipopolysaccharide in mouse **dendritic cells**.
AUTHOR(S): Ai, Huachang; Yu, Yinni; Zhang, Minghui; Xu, Hongmei; Qi, Runxi; Yan, Nianyi; Liu, Shuyan; Wang, Wenya; Guo, Zhenghong; Guo, Jun; Qin, Zhihai; Cao, Xuetao (1)
CORPORATE SOURCE: (1) Institute of Immunology, Second Military Medical University, 800 Xiangyin Road, Shanghai, 200433: caoxt@public3.sta.net.cn China
SOURCE: Immunology, (May, 2002) Vol. 106, No. 1, pp. 38-45. http://www.blackwell-science.com/cgi-bin/jnlpage.asp?[illegible] ISSN: [illegible].
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB [illegible] Toll-like receptor (TLR) [illegible] capable of recognizing pathogen-associated molecular patterns [illegible] such as lipopolysaccharide (LPS) and bacterial **oligonucleotides** (CpG ODN). TLR2 and TLR4 are major receptors for Gram-positive and Gram-negative bacterial cell wall components, respectively. TLR9 is necessary for CpG signalling. LPS or CpG ODN can activate immature **dendritic cells** (DC) and induce DC maturation characterized by production of cytokines, up-regulation of co-stimulatory molecules, and increased ability to activate T cells. However, little is known regarding the regulation of TLR gene expression in mouse DC. In this study, we investigated the regulation of TLR2, TLR4 and TLR9 gene expression by LPS in murine immature DC. TLR2, TLR4 and TLR9 mRNA were up-regulated following LPS stimulation. The up-regulation of TLR9 expression coincided with significantly increased production of tumour necrosis factor-alpha induced by LPS plus CpG ODN. While inhibition of extracellular signal-related kinase and NF-kappaB activation suppressed the up-regulation of the expression of TLR2, TLR4 and TLR9 mRNA, inhibition of p38 kinase prevented the up-regulation of TLR2 and TLR4 mRNA expression but [illegible] the up-regulation of TLR9 expression. These results [illegible] TLR2, TLR4 and TLR9 gene expression [illegible] immature DC. The up-regulation of TLR2, TLR4 and TLR9 [illegible] the overall responsiveness [illegible] to explain the synergy between LPS and [illegible] in the induction of cytokine production.

119 ANSWER [illegible] BIOSIS [illegible] BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Regulation of Nf-kappa-B pathway genes in **dendritic cells** [illegible] macrophages assessed by gene expression profiling.
AUTHOR(S): Faltarkis, Ioannis [1] ; Alcantara, Orlando [1]; Boldt, David H. [1]
CORPORATE SOURCE: [1] Medicine/Hematology, University of Texas Health Science Center, San Antonio, TX USA
SOURCE: Blood, [illegible], [illegible], [illegible]. [illegible]. [illegible].
[illegible]
DOCUMENT TYPE:
LANGUAGE:
SUMMARY LANGUAGE:

AB NFkappa-B/Rel transcription factors (mainly c-Rel, RelB, and p50) have been implicated in the differentiation of monocytes to either DCs or macrophages, as well as in the maturation of DCs from an immature to antigen-presenting cells. Recent studies of the expression pattern of Rel proteins and their inhibitor IkappaBs suggest that their activity during this differentiation process is transcriptional. To investigate differential gene expression between macrophages and DCs, we used commercially available human microarrays (GEArray™ Kit), which contained 4 of the NFkappa-B/Rel family genes (p65, c-Rel, RelB, and p50) and 32 additional genes that are known to be under transcriptional control of NFkappa-B. Peripheral blood monocytes, human adherent peripheral blood [illegible] and [illegible] (IL-10 + TNF-beta + IL-4 respectively) [illegible]. [illegible] cytokines, macrophages were treated with [illegible] for the last 24 hours of culture to induce maturation. Cells were harvested after 7 days, cDNA was prepared and radiolabeled with alpha-32P-dCTP, then hybridized to gene arrays containing specific gene probes. beta-actin and GAPDH or PUC18 **oligonucleotides** served as positive or negative controls, respectively. The expression of all 4 NFkappa-B/Rel family genes examined was significantly up-regulated in maturing DCs compared to macrophages. The strongest difference was observed for c-Rel. Sequential RT-PCR determinations of c-Rel, RelB, and p50 mRNAs confirmed these observations. Stimulation of macrophages with LPS resulted in induction of the same genes, but the expression of c-Rel remained higher in DCs. Among the 32 NFkappa-B/Rel target genes, 11 were consistently up-regulated in mature DCs compared to macrophages. These genes were I-kappa-B-alpha, NIK (serine/threonine protein kinase), ICAM-1, P-selectin, TNFR2, TNFAIP3 (tumor necrosis factor alpha-induced protein 3), IL-1alpha, IL-1R1, IL-1R2, IRAK (IL-1 receptor-associated kinase), and TANK (TRAF family member-associated NFkappa-B activator). By contrast, only mgl-1 (monocyte [illegible] macrophages. Results [illegible] experiments. Genes whose expression [illegible] included c-myc, VCAM-1, E-SEL, MIP-3B, IL-6, IL-8, and IL-10. We conclude that NFkappa-B/Rel family genes, especially c-Rel, are selectively induced during **dendritic cell** maturation. Moreover, this process is associated with expression of a unique subset of genes that are transcriptionally targeted by NFkappa-B/Rel factors.

L19 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: [illegible]:542[illegible] CAPLUS
DOCUMENT NUMBER: 137:[illegible]
TITLE: Potential role of phosphatidylinositol 3 kinase, rather than DNA-dependent protein kinase, in dsRNA-induced innate antiviral [illegible]
AUTHOR(S): Ishii, Ken J.; Takeshita, Fumihiko; Gursel, Ihsan; Gursel, Mayda; Conover, Jacqueline; Klinman, Dennis; Akira, S.
CORPORATE SOURCE: [illegible] Vaccine Research, [illegible], Bethesda, MD, [illegible] Institute, Osaka, Japan
SOURCE: [illegible] Immunology (Baltimore), [illegible], 167(2), [illegible]
[illegible]
CODEN: JIMMAA; ISSN: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB [illegible] dsRNA, a potent inducer of innate immune responses. Here we demonstrate that DNA-dependent protein kinase (DNA-PK) mediates dsRNA signaling. Specifically, wortmannin, an inhibitor of phosphatidylinositol 3 kinase (PI3-kinase) including DNA-PK, interferes with dsRNA-dependent cell activation, and DNA-PK mutant mice fail to respond to dsRNA stimulation. [illegible]

[illegible] with
[illegible] preferentially
[illegible] NF-.kappa.B activity. We find that
DNA-PK is not [illegible], since three strains of DNA-PK KO
mice responded [illegible] DNA. These results support a model in
which [illegible] is dependent on TRAF-6 but not DNA-PK, and suggest
that [illegible] members of the PIK-kinase family play a crit.
[illegible] DNA to TLR-4.
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:499249 CAPLUS
DOCUMENT NUMBER: 135:71280
TITLE: Activation and inhibition of the immune system
INVENTOR(S): Farrell, Brian; Fellmann, Marc
PATENT ASSIGNEE(S): The Mathilda and Terence Kennedy Institute of
Rheumatology Trust, UK
SOURCE: PCT Int. Appl., [illegible] pp.
[illegible]
DOCUMENT TYPE: Patent
LANGUAGE: [illegible]
FAMILY ACC. NUM. [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001003748	A	20010118	WO 2000-GB4925	20001222
WO 2001003748	A2	20020117		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1244466	A2	[illegible]	[illegible]	[illegible]
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, SE, MC, PT, IE, FI, [illegible]				

PRIORITY APPLN. INFO.: [illegible]

AB [illegible] NF-.kappa.B activity, induction of an
[illegible] NF-.kappa.B inhibitor and the inhibition and
activation of [illegible] by the administration of an activator or
inhibitor of NF-.kappa.B is described. Examples of NF-.kappa.B inhibitors
include I.kappa.B.alpha., I.C, a single DNA sequence encoding
I.kappa.B.alpha., anti-sense oligonucleotides against NF-.kappa.B
sequences, anti-sense RNA, and anti-NF-.kappa.B antibodies. Examples of
NF-.kappa.B inducers include NIK, MEKK, IKK2, TRAF2 and Bcl 3. Also
disclosed are methods for identifying inducers and inhibitors of NF-.kappa.B, for
example adenoviral vectors.

L19 ANSWER 5 OF 11 CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
[illegible]
[illegible]
INVENTOR(S): [illegible]

L.; Mataraza, Kevin N.; Williams, I. Mikey; Wood, William L.; Yamazaki, Kaoru S.

PATENT ASSIGNEE(S): [illegible], Inc., USA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: [illegible]

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

WO 2000053752	A2	20000914	WO 1999-US31274	19991230
WO 2000053752	A3	20001221		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

WO [illegible]	[illegible]	[illegible]	[illegible]	[illegible]
WO [illegible]	[illegible]	[illegible]		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

WO [illegible]	[illegible]	[illegible]	[illegible]	[illegible]
WO [illegible]	[illegible]	[illegible]		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

W [illegible]	[illegible]	[illegible]	[illegible]	[illegible]

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

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[illegible]
RW: GH, GM, KE, LS, [illegible]
KZ, MD, RU, TJ, TM, AT, BE, CH, CY, [illegible]
IE, IT, LU, MC, NL, PT, SE, TR, [illegible]
GW, ML, MR, NE, SN, TD, TG

AU 2001071973 A[illegible] [illegible]
US 2002090681 A1 [illegible]

PRIORITY APPLN. INFO.:

[illegible]
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[illegible]
[illegible]
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[illegible]
[illegible] W 20000321
[illegible] W 20000602
US [illegible]-215667P P 20000622
[illegible]-644547 A 20000822
WO 2000-US23328 W 20000824
US 2000-242837P P 20001024
WO 2000-US30873 W 20001110
US 2000-253646P P 20001128
WO 2000-US32678 W 20001201
US 1998-85579P P 19980515
US 1998-112514P P 19981215
US 1998-113300P P 19981222
US 1998-114[illegible]P P [illegible]
US 1998-11[illegible]P P [illegible]
US 1998-11[illegible]P P [illegible]
US 1998-114190P P [illegible]
[illegible]
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[illegible]
[illegible] P 19990310
[illegible] P 19990312
[illegible]P P 19990323
[illegible]P P 19990323
[illegible]P P 19990323
[illegible]626P P 19990324
[illegible] P 19990329
[illegible]P P 19990331
[illegible]
US 1999-128849P P 19990412
[illegible] P 19990413
WO 1999-US8615 W 19990420
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[illegible] A 20001108
WO [illegible] W 20001108
US [illegible] A 20001220
WO [illegible] W 20010308
[illegible] A [illegible]
[illegible] A [illegible]
[illegible] W [illegible]
[illegible] W [illegible]
[illegible] A [illegible]
[illegible] A [illegible]
[illegible] A [illegible]
[illegible] A [illegible]
[illegible] A [illegible]
[illegible] A [illegible]
[illegible] A 20010525
[illegible] W 20010601

AB Proteins that are antagonists of the biological interleukin 17 and that therefore may be of use in the treatment of diseases associated with immune dysfunction are described. Cloning and expression vectors and host cells, fusion proteins, antibodies to the proteins, and methods of manufg. them are described. The genes for these proteins are expressed in a wide array of tissues. The proteins do not bind known interleukin 17 receptors but one of the proteins was identified as a receptor for two of the others. The proteins show interleukin 17-like activities, such as activation of NF-.kappa.B.

L19 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:24211 CAPLUS
DOCUMENT NUMBER: 134:95602
TITLE: Antisense **oligonucleotide** [illegible] of BAFF expression
INVENTOR(S): Baum, Brenda J.; [illegible]
PATENT ASSIGNEE(S): [illegible], U.S., USA
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT:
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6171832	B1	[illegible]	US [illegible]	19991130
WO 2001034827	A1	20010517	WO [illegible]	20001103

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

[illegible] A [illegible]
[illegible] A [illegible]

AB Antisense **oligonucleotides**, [illegible], which hybridize to [illegible] are [illegible]

nuclear factor-kappa B, [illegible]

[illegible] - [illegible] **dendritic cell** [illegible]

oligonucleotides, [illegible] BAFF.

oligonucleotides

REFERENCE COUNT: [illegible] REFERENCES AVAILABLE FOR THIS RECORD. ALL REFERENCES AVAILABLE IN THE RE FORMAT

L19 ANSWER [illegible] OF 11 CABA COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: A toll-like receptor recognizes bacterial DNA. [Erratum to document cited in CA134:161862]
AUTHOR(S): Hemmi, Hiroaki; Takeuchi, Osamu; Kawai, Taro; Kaisho, Tsuneyasu; Sato, Shintaro; Sanjo, Hiroaki; Matsumoto, Makoto; Hoshino, Katsuaki; Wagner, Hermann; Takeda, Kiyoshi; Akira, Shizuo
CORPORATE SOURCE: Department of Host Defense, Research Institute for Microbial Diseases, Osaka University and Core Research for Evolutional Science and Technology, Osaka, [illegible], Japan
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB The [illegible].

L19 ANSWER [illegible] OF 11 CABA COPYRIGHT 2004 ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Expression of different NF-.kappa.B pathway genes in dendritic cells [DCs] in macrophages assessed by gene expression profiling
AUTHOR(S): Baltathakis, Ioannis; Alcantara, Orlando; Boldt, David H.
CORPORATE SOURCE: Medicine/Hematology, University of Texas Health Science Center, San Antonio, TX, [illegible], USA
SOURCE: Journal of [illegible], [illegible], [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB NF-.kappa.B/Rel transcription factors play a central role in the differentiation of dendritic cells from [illegible]. Recent studies at the cellular level have pointed to inhibitors (I.kappa.Bs) suggest that these proteins regulate this differentiation process is [illegible]. To comparatively analyze differential gene expression between [illegible], the authors used commercially available gene microarrays (GEArray Kit, [illegible]) that contain probes for the NF-.kappa.B/Rel family genes (p105/p50, p100/p52, RelB, c-Rel, and v-Rel). Genes either in the NF-.kappa.B signal transduction pathway or under transcriptional control of NF-.kappa.B/Rel factors. [illegible] adherent peripheral blood monocytes [illegible] IL-4 [illegible] for 3 days. [illegible] treated with lipopolysaccharide [illegible] in the presence of IL-4 [illegible] for 3 days. [illegible] with [illegible] and [illegible] specific [illegible] oligonucleotides [illegible]

similarly upregulated in mature DCs compared to macrophages. The strongest differences were seen for c-rel. NF-ΚB genes, c-rel, RelB, and p105 mRNAs confirmed these observations. Among the 32 NF-.kappa.B/Rel pathway genes, 14 were upregulated in mature DCs compared to macrophages. These genes were I.kappa.B.alpha., IKK-.beta., MIF, ICAM-1, P-selectin, E-selectin, TNF-.alpha., TNFR2, TRAIP, IL-1.alpha., IL-1R1, IL-1RL, IRAK, and TANK. By contrast, fully mature monocyte-derived [illegible] was upregulated in macrophages compared to DCs. NF-.kappa.B pathway genes upregulated in DCs compared to macrophages were similarly expressed in monocytes that were induced to differentiate into DCs. [illegible] macrophages. The [illegible] NF-.kappa.B pathway genes, such as c-rel, are specifically expressed at high levels in differentiating monocytes towards DCs. Moreover, [illegible] with activation of different NF-.kappa.B signal transduction pathways in DCs and macrophages and with [illegible] DCs that are transcriptionally regulated by NF-.kappa.B/Rel factors. The results illustrate the ability of the NF-.kappa.B pathway to respond to different [illegible] by activating in a cell-specific manner unique signaling pathways and subsets of NF-.kappa.B target genes.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:904923 CAPLUS

DOCUMENT NUMBER: 134:161862

TITLE: A Toll-like receptor recognizes bacterial DNA

AUTHOR(S): Hemmi, Hiroaki; Takeuchi, Osamu; Kawai, Taro; Kaisho, Tsuneyasu; Sato, Shintaro; Sanjo, Hiroaki; Matsumoto, Kenji; Hoshino, Kazuhiko; Wagner, Hermann; Takeda, Kiyoshi; Akira, Shizuo

CORPORATE SOURCE: [illegible]

SOURCE: [illegible], 408(6813), 740-745
CODEN: NATURS; ISSN: 0028-0836

PUBLISHER: [illegible]

DOCUMENT TYPE: Journal

LANGUAGE: [illegible]

AB DNA from bacteria but not vertebrates activates mammalian immune cells, which express a Toll-like receptor. Unmethylated CpG dinucleotides in the bacterial DNA. In contrast, mammalian DNA has a low frequency of CpG dinucleotides, and these are mostly methylated; therefore, mammalian DNA does not have immune-stimulatory activity. CpG DNA induces a strong T-helper-1-like inflammatory response. Accumulating evidence has revealed the therapeutic potential of CpG DNA as adjuvants for [illegible] therapy, allergy and infectious diseases. Despite the promising [illegible], the mol. mechanism by which CpG DNA activates [illegible] Here the authors show that [illegible] Toll-like receptor 9 (TLR9). TLR9-[illegible] dendritic cells. [illegible] TLR9-[illegible] CpG DNA from [illegible]

PREPARED [illegible] THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS [illegible] ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER [illegible]

ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE:
dendritic cells
AUTHOR(S):
CORPORATE SOURCE:
Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA
SOURCE:
Molecular Therapy (2000), 1(5, Pt. 1), 430-437
CODEN: MTOHCK; ISSN: 1525-0016
PUBLISHER:
Academic Press
DOCUMENT TYPE:
Journal
LANGUAGE:
English
AB **Dendritic cells** (DC) classically promote immune responses but can be manipulated to induce antigen-specific hyporesponsiveness in vitro. The expression of costimulatory mols. (CD54, CD86, CD80) at the DC cell surface correlates with their ability to induce or suppress immune responses. Expression of these mols. is assocd. with NF-.kappa.B-mediated transcriptional events. DC [illegible] NF-.kappa.B-dependent [illegible] to the nucleus. To this end, we used 'decoy' double-stranded [illegible] NF-.kappa.B [illegible] NF-.kappa.B-dependent transcription of a reporter gene. Moreover, [illegible] **oligonucleotide** decoys [illegible] lipopolysaccharide (LPS)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B ODN selectively suppressed the cell-surface expression of costimulatory mols. without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B ODN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B ODN-modified bone marrow-derived [illegible] allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B [illegible] involved in immune [illegible] one means to promote [illegible] 2000 Academic Press.
REFERENCE COUNT:
[illegible] THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS [illegible] IN THE RLE FORMAT]
L14 ANSWER [illegible]
ACCESSION NUMBER:
TITLE:
oligonucleotides [illegible] nuclear factor .kappa.B :
[illegible]-mediated hyporesponsiveness.
AUTHORS:
CORPORATE SOURCE:
SOURCE:
COUNTRY:
DOCUMENT TYPE:
FILE SEGMENT:
LANGUAGE:

SUMMARY LANGUAGE: English

AB Background. DNA containing the CpG motif is associated with immunomodulation of the innate immune response. Preexposure of macrophages to CpG DNA elicits a hyporesponsiveness to subsequent lipopolysaccharide (LPS) stimulation. We tested the hypothesis that this effect is due to decreased nuclear translocation of **nuclear factor** **kappa.B** (NK-.kappa.B). Methods. Murine macrophage-like RAW 264.7 cells were incubated with [illegible] **oligonucleotides** [illegible] restimulation with [illegible] LPS [illegible]. The [illegible] cotransfected with an NF-.kappa.B [illegible] and a [illegible] As appropriate, [illegible] stimulated [illegible] I-.kappa.B by Western [illegible], [illegible] activity, [illegible], c-[illegible] N-terminal kinase, [illegible] activity assay. Results. NF-.kappa.B [illegible] was decreased as demonstrated by [illegible] activity assay in the [illegible] CpG ODN pretreatment groups. Unlike endotoxin, [illegible] CpG DNA preexposure increased cytoplasmic phospho-I.kappa.B.alpha. and did not abrogate mitogen-activated protein kinase activity. Conclusions. In macrophages, exposure to CpG DNA increases expression of the inhibitory p50 NF-.kappa.B homodimer and decreases NF-.kappa.B activity without inhibition of I.kappa.B kinases. Mitogen-activated protein kinase activity remains intact. Understanding these interactions between different toll receptor ligands may provide insight into novel therapeutic modalities.

L21 ANSWER 1 OF 1 [illegible]

ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] dendritic cells.
AUTHOR(S): [illegible]; Yu, Yuanli; Zhang, Minghui; Xu, Hongmei; Qi, Huimin; Yan, Yinyin; Liu, ShuKun; Wang, Wenya; Guo, Zhongmin; Bao, Yan; Wan, Xuehai; Cao, Xuetao (1)
CORPORATE SOURCE: (1) Institute of Immunology, Second Military Medical University, 800 Xiangyin Road, Shanghai, 200433: caoxtapublic@sta.net.cn China
SOURCE: Immunology, (May, 2004) Vol. 112, No. 1, pp. 36-45. http://www.blackwell-science.com/cgilib/jnlpage.asp?journal=imm&file=imm.htm. ISSN: 0019-2805.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Toll-like receptors (TLR) are seminal receptors capable of recognizing pathogen-associated molecule patterns (PAMP) such as lipopolysaccharide (LPS) and CpG-containing **oligonucleotides** (CpG ODN). TLR2 and TLR4 are [illegible] and Gram-positive bacteria. Cell wall lipopeptides, a major PAMP, [illegible] necessary for CpG signalling. [illegible] **dendritic cells** [illegible] by production of cytokines, [illegible] by cytokines, and increased ability to activate T cells. However, little is known regarding the regulation of TLR gene expression in DC. In this study, we investigated the regulation of TLR2, TLR4 and TLR9 gene expression by LPS in murine immature DC. TLR2, TLR4 and TLR9 mRNA were up-regulated following LPS stimulation. The up-regulation of TLR9 expression coincided with significantly increased production of tumour necrosis factor-alpha induced by LPS plus CpG ODN. While inhibition of extracellular signal-related kinase and NF-kappaB activation suppressed the up-regulation of the expression of TLR2, TLR4 and TLR9 mRNA, inhibition of p38 kinase prevented the up-regulation of TLR2 and TLR4 mRNA expression but enhanced the up-regulation of TLR9 expression. These results demonstrate that TLR2, TLR4 and TLR9 gene expression was differently regulated by LPS in murine immature DC. Up-regulation of TLR2, TLR4 and TLR9 expression by LPS might [illegible] the overall responses [illegible] between [illegible] by [illegible] pr[illegible].

L21 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] dendritic cells [illegible].
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible], University of Texas Health Science [illegible], Texas [illegible], USA.
CONTRACT NUMBER: [illegible]
SOURCE: JOURNAL OF [illegible] BIOCHEMISTRY, [illegible] 1-[illegible] 2[illegible].
[illegible]
PUB. COUNTRY: [illegible]
DOCUMENT TYPE: [illegible]; [illegible]
LANGUAGE: [illegible]
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY DATE: [illegible]

AB NF-kappaB/Rel transcription factors have been implicated in the differentiation of myeloid cells, including **dendritic cells** (DCs), or macrophages, as well as in the maturation of DCs from antigen-processing to antigen-presenting cells. Recent studies of the expression pattern of Rel proteins and their inhibitors (IkappaBs) suggest that their regulation during this differentiation process is transcriptional. To investigate differential gene expression between macrophages and DCs, we used commercially available nylon microarrays (GEArray KIT, which included genes for the NF-kappaB/Rel family and IkappaB family, p105/p50, p100/p52, RelA, RelB, c-Rel, and IkappaBalpha, IkappaBbeta, IkappaBepsilon, IkappaBgamma, IkappaBzeta, Bcl-3, and IkappaBNS) to the NF-kappaB signal transduction pathway. Human monocytes were purified by [illegible] and cultured with GM-CSF + IL-4 for 6 days (immature DCs) or with GM-CSF + IL-4 + TNF-alpha for 6 days (mature DCs). For macrophages, monocytes were cultured with GM-CSF for the last 48 h of culture to induce maturation. After 6 days, cDNA was prepared and radiolabeled with alpha-[32]P-dCTP, then hybridized to gene arrays containing specific gene probes. Beta-actin and GAPDH or PUC18 **oligonucleotides** were used as positive or negative controls, respectively. The expression of all four NF-kappaB/Rel family genes examined was significantly upregulated in maturing DCs compared to macrophages. The greatest difference was observed for c-rel. RT-PCR determinations of c-rel, RelB, and p105 mRNAs confirmed these observations. Among the 32 NF-kappaB/Rel pathway genes, 14 were upregulated in mature DCs compared to macrophages. These genes were IkappaBalpha, IKK-beta, NIK, ICAM-1, P-selectin, E-selectin, TNF-alpha, TNFR2, TNFAIP3, IL-1alpha, IL-1R1, IL-1R2, IRAK, and TANK. By contrast, only mcp-1 (monocyte chemotactic protein 1) was upregulated in macrophages compared to DCs. NF-kappaB pathway genes upregulated in DCs compared to macrophages were constitutively expressed in monocytes then selectively downregulated during macrophage but not DC differentiation. The distinct [illegible] expression of these genes in maturing DCs and macrophages [illegible] NF-kappaB/Rel family [illegible], [illegible] Rel proteins. Thus, this differential expression [illegible] with activation of different NF-kappaB signal transduction pathways in DCs and macrophages and with expression of a unique subset of NF-kappaB/Rel genes that are transcriptionally targeted by NF-kappaB/Rel proteins. The results illustrate the ability of the NF-kappaB pathway to respond to differentiation stimuli by activating in a cell-specific manner unique signaling pathways and subsets of NF-kappaB target genes.

L21 ANSWER 3 OF 15 MEDLINE [illegible]
ACCESSION NUMBER: 2000410045 MEDLINE
DOCUMENT NUMBER: [illegible] PubMed ID: [illegible]
TITLE: [illegible] **dendritic cells** [illegible] with NF-kB [illegible] [illegible].
COMMENT: [illegible]
AUTHOR: [illegible]
[illegible]
SOURCE: [illegible]
PUB. [illegible]
[illegible]

LANGUAGE: [illegible]
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY WEEK: [illegible]

AB Dendritic cells [illegible] hyperactivity [illegible] (CD54, CD80, [illegible]) to initiate and regulate immune responses. Expression of these molecules is associated with NF-kB-dependent transcription of their genes. DC tolerogenicity has been associated with impaired NF-kB-dependent transcription of costimulatory genes as well as NF-kB translocation to the nucleus. In this report, we demonstrate that double-stranded oligodeoxyribonucleotides containing binding sites for NF-kB (NF-kB ODN) are efficiently incorporated by bone marrow-derived DC and specifically inhibit NF-kB-dependent transcription of a reporter gene. Moreover, exposure of DC to the **oligonucleotide** decoys inhibited lipopolysaccharide (LPS)-induced nitric oxide production, a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-kB ODN selectively suppressed the cell-surface expression of costimulatory molecules without interfering with MHC class I or class II expression. Furthermore, NF-kB [illegible] and this was associated with [illegible]. Finally, infusion of NF-kB [illegible] prolongation of allograft survival [illegible]. Specific interference with NF-kB [illegible] in immunogenicity. Specific [illegible] to promote tolerance [illegible].

L21 ANSWER 4 OF 10 MEDLINE
ACCESSION NUMBER: 2002139184 MEDLINE
DOCUMENT NUMBER: 21864248 PubMed ID: 11773067
TITLE: Molecular mechanisms involved in CD43-mediated apoptosis of TF-1 cells. Roles of transcription Damx expression, and adhesion molecules.
AUTHOR: Cermak Lukas; Simova Darka; [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUB. [illegible]
[illegible]
LANGUAGE: [illegible]
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY WEEK: [illegible]

AB [illegible] dendritic cells. [illegible] anti-CD43 [illegible] induce apoptosis [illegible] it also triggers apoptosis [illegible] TF-1. The kinetics [illegible] with the first [illegible]

[illegible] anti-CD43 (MEM-59) or anti-CD99 mAb. The MEM-induced apoptosis of TF-1 cells was also inhibited by the [illegible]. MEM-mediated apoptosis was preceded by the suppression of the DNA binding activity of the transcription factor AP-1. DNA array screening revealed that the expression of several genes encoding apoptosis-regulating proteins, including 14-3-3 proteins and the granulocyte macrophage colony-stimulating factor (GM-CSF) receptor beta-subunit, was suppressed in TF-1 cells bound to immobilized MEM-59. The down-regulation of 14-3-3 proteins and GM-CSF receptor beta was accompanied by translocation of the proapoptotic protein Bad to the mitochondria. These results suggest that engagement of CD43 may, presumably via [illegible] the repressing transcription, initiate a Bad-dependent apoptotic pathway.

L21 ANSWER 5 OF 15 [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] via peptidoglycan and lipoteichoic acid in Gram-positive bacteria: [illegible] Toll-like receptor 2.
AUTHOR: [illegible] Jeffrey B; Brunn Gregory J; Kodaira Yuzo; Platt John L
CORPORATE SOURCE: Department of Immunology, Mayo Clinic, 2-66 Medical Sciences Building, Rochester, MN 55905, USA.
CONTRACT NUMBER: [illegible] NIDR
HL 52397 (NHLBI)
SOURCE: JOURNAL OF IMMUNOLOGY, (2002 May 15) 168 (10) 5233-9. Journal code: [illegible]. ISSN: [illegible].
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Last Updated in [illegible]:

AB [illegible] dendritic cells, [illegible] molecules is not known. We [illegible] dendritic cells [illegible] fragments of heparan sulfate [illegible]. Dendritic cells [illegible] as measured by costimulatory protein expression, [illegible], and T lymphocyte stimulation, but this maturation was absent when Toll-like receptor 4 was mutated or inhibited. These findings suggest that Toll-like receptors in mammals may monitor tissue well-being by recognizing fragments of endogenous matrix molecules.

L21 ANSWER 6 OF 15 MEDLINE
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] dendritic cells [illegible]
[illegible]
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible]
[illegible]
[illegible]
SOURCE: [illegible] Suppl 31-2... Ref:

[illegible]

AB **Dendritic cells** [illegible] constitute a complex system of uniquely well-equipped antigen-presenting cells that initiate and regulate immune responses. Extensive recent studies have improved our understanding of DC development, differentiation, activation, and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and biological functions. These factors may determine the T-cell polarizing signals and type of T cell response (T helper 1, T helper 2, or T regulatory) induced by DC. Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T-regulatory cell activity, and programmed death of activated T cells. [illegible] emerging information suggests that [illegible] expression of death-inducing ligands [illegible], microenvironmental factors (including cytokines, such as immunosuppressive cytokines), and inhibition of nuclear factor kappa B pathway proteins (e.g., nuclear factor-kappaB) can impart tolerogenic properties to DC. Manipulation of DC by control of their maturation and differentiation, or genetic engineering of these cells to express immunosuppressive molecules, offers potential for therapy of allograft rejection and autoimmune disease. In this brief overview, we outline principles and methods for generation of "tolerogenic" DC and outcomes that have been reported in experimental models. Space constraints limit literature citations.

L21 ANSWER 7 OF 15 MEDLINE
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible] PubMed ID: [illegible]
TITLE: [illegible] **dendritic cells** [illegible]

AUTHOR: [illegible]

CORPORATE SOURCE: [illegible]

SOURCE: [illegible]

PUB. TYPE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY DATE: [illegible]

AB We systematically [illegible] levels of **dendritic cells** [illegible] phenotype, [illegible]

CD86) and numerous cytokines and chemokines [illegible] (e.g., Flt-3L, G-CSF, IL-1alpha and -1beta, IL-[illegible], IL-12, [illegible], and -22, MIP-2, and [illegible]) and were significantly better at [illegible] effector T cell responses in vitro. Furthermore, mice [illegible] with tumor peptide-pulsed mature DCs [illegible] with a weakly immunogenic [illegible] maturity [illegible]

L21 ANSWER [illegible] CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]:548719 CAPLUS
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] role of phosphatidylinositol 3 kinase, rather than DNA-dependent protein kinase, in CpG DNA-induced immune activation
AUTHOR(S): Ishii, Ken J.; Takeshita, Fumihiko; Gursel, Ihsan; Gursel, Mayda; Conover, Jacqueline; Nussenzweig, Andre; Klinman, Dennis M.
CORPORATE SOURCE: Section of Retroviral Immunology, Division of Viral Products, Center for Biologics Evaluation and Research, Food and Drug Administration, National Institutes of Health, Bethesda, MD, 20892, USA
SOURCE: Journal of Experimental Medicine [illegible], [illegible]-274
[illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]

AB [illegible] stimulate a strong innate immune [illegible] that DNA-dependent protein kinase (DNA-PK) mediates [illegible] signaling. Specifically, wortmannin (an inhibitor of phosphatidylinositol 3 kinase [PI3]-kinases including DNA-PK) interferes with CpG-dependent cell activation, and DNA-PK knockout (KO) mice fail to respond to CpG stimulation. Current studies establish that wortmannin actually inhibits the uptake and colocalization of CpG DNA with toll-like receptor (TLR)-9 in endocytic vesicles, thereby preventing CpG-induced activation of the NF-.kappa.B
signaling cascade. We find that DNA-PK is not [illegible], since three strains of DNA-PK KO mice responded normally to [illegible]. These results support a model in which [illegible] TLR-9 but not DNA-PK, and suggest that [illegible] the PI3-kinase family [illegible].

REFERENCE COUNT: [illegible] THERE ARE [illegible] REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO. KIND DATE APPLICATION NO. DATE

WO [illegible]	A[illegible]	[illegible]	[illegible]	[illegible]
WO [illegible]	A[illegible]	[illegible]	[illegible]	[illegible]

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

[illegible]

PRIORITY APPLN. INFO.: [illegible] A 19991224
[illegible] W 20001222

AB [illegible] by **NF-.kappa.**
B inducers, [illegible] by **NF-.**
kappa.B [illegible] and activation of immune response by the administration of an activator or inhibitor of **NF-.kappa.B** is disclosed. Examples of **NF-.kappa.B** inhibitors include I.kappa.B.alpha., PSI, a nucleotide sequence encoding I.kappa.B.alpha., anti-sense nucleic acid encoding an **NF-.kappa.**
B sequence, such as Rel B, and anti-**NF-.kappa.**
B antibodies. Examples of **NF-.kappa.B** inducers include NIK, MEKK, IKK2, TFRRP and Rel B. Also disclosed are vectors encoding inducers and inhibitors of **NF-.kappa.**
B, for example alkan viral vectors.

L21 ANSWER [illegible] OF [illegible] [illegible] COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] by sequence [illegible] and their use in treatment of immune [illegible]
INVENTOR(S): [illegible]; Elliott, Ellen; Fong, Sherman; Goddard, Audrey; Jakubowski, Paul J.; Grimaldi, Christopher J.; Kinney, Austin L.; Li, Hamburg; Hillan, Kenneth J.; Lukas, Tahiri; Van, Lookeren Menno; Vandlen, Richard L.; Watanabe, Colin K.; Williams, P. Mickey; Wood, William I.; Yansura, Daniel G.
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

[illegible]

W: [illegible]

RW: [illegible]

WO [illegible]
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SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
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EP 1240329 AC [illegible] FI [illegible]
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TG
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WO 2002056284 A2 20020718 WO 2001-US21795 20010709
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RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AT [illegible]
[illegible]
PRIORITY [illegible]

NF-.kappa.B.

L21 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **oligonucleotide** [illegible] RANK expression
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6171860	B1	[illegible]	[illegible]	[illegible]
WO 2001034757	A2	[illegible]	[illegible]	[illegible]

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP [illegible] A1 [illegible] EP 2000-974012 20001030

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL

PRIORITY APPLN. INFO.: US 1999-452096 A 19991109
WO 2000-US29818 W 20001030

AB Antisense **oligonucleotides**, compns., and methods are provided for inhibiting the expression of RANK (receptor activator of nuclear factor-kappa B), a receptor known to enhance T-cell growth and **dendritic cell** function. The compns. comprise antisense compds., particularly antisense **oligonucleotides**, that hybridize to nucleic acids encoding RANK. The [illegible] **oligonucleotides** [illegible]

RE.CNT [illegible] THERE ARE [illegible] RE FORMAT

L21 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] expression during [illegible]
dendritic cells [illegible]
oligonucleotide [illegible]
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUBLISHER: [illegible]

Biology

DOCUMENT TYPE:
LANGUAGE:

AB **Dendritic cells**
oligonucleotide
oligonucleotide

REFERENCE COUNT:

L21 ANSWER
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE: A Toll-like receptor recognizes bacterial DNA. [Erratum to document cited in CA134:161862]
AUTHOR(S): Hemmi, Hiroaki; Takeuchi, Osamu; Kawai, Taro; Kaisho, Tsuneyasu; Sato, Shintaro; Sanjo, Hideki; Matsumoto, Makoto; Hoshino, Katsuaki; Wagner, Hermann; Takeda, Kiyoshi; Akira, Shizuo
CORPORATE SOURCE:
SOURCE:
PUBLISHER:
DOCTYPE:
LANGUAGE:
AB

L21 ANSWER
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE:
AUTHOR(S):
CORPORATE SOURCE:
SOURCE:

PUBLISHER:
DOCUMENT TYPE:
LANGUAGE:
AB [illegible] dependent on the presence of unmethylated CpG dinucleotides in the bacterial DNA. In contrast, mammalian DNA has a low frequency of CpG dinucleotides, and these are mostly methylated; therefore, mammalian DNA does not have immuno-stimulatory activity. CpG DNA induces a [illegible] inflammatory response. Accumulating evidence has revealed the therapeutic potential of CpG DNA as adjuvants for vaccination strategies for cancer, allergy and infectious diseases. [illegible] dendritic cells. [illegible] Low lethal effect of CpG DNA [illegible] pro-inflammatory cytokine levels. The [illegible] Th1-type response was also abolished in [illegible]. Thus, vertebrate immune systems appear to have evolved a specific [illegible] that distinguishes bacterial DNA from self-DNA.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 15 OF 15 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2002330736 EMBASE
TITLE: Preexposure of murine macrophages to CpG-containing **oligonucleotides** results in nuclear factor .kappa.B p50 homodimer-associated hyporesponsiveness.
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
COUNTRY:
DOCUMENT TYPE:
FILE SEGMENT: [illegible] Pathological Anatomy
LANGUAGE:
SUMMARY LANGUAGE:
AB Background. DNA [illegible] the CpG motif is associated with immunomodulation of the innate immune response. Preexposure of macrophages to CpG DNA elicits a hyporesponsiveness to subsequent lipopolysaccharide (LPS) stimulation. We tested the hypothesis that this effect is due to decreased nuclear translocation of nuclear factor .kappa.B (NF-.kappa.B). Methods. Murine macrophage-like RAW 264.7 cells were incubated with 1 .mu.g/mL CpG-containing **oligonucleotides** [illegible] hours followed by restimulation with 1 .mu.g/mL LPS. Nuclear extracts of cells were [illegible] with an NF-.kappa.B sensitive [illegible]. Cytoplasmic [illegible] NF-.kappa.B [illegible] NF-.kappa.B. [illegible] NF .kappa.B [illegible]

expression of the inhibitory p NF-.kappa.B
homodimer and wa NF-.kappa.B
without inhibiting

L23 ANSWER 1 OF [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **dendritic cells** [illegible]
[illegible] in transplantation and autoimmune [illegible].
AUTHOR(S): [illegible]; [illegible]; Thomson, Angus W.
CORPORATE SOURCE: [illegible] Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, 200 Lothrop Street, E1554, Biomedical Science Tower, Pittsburgh, PA, 15213: lui@tmx.upmc.edu USA
SOURCE: Transplantation (Baltimore), (January 15, 2002) Vol. 73, No. 1 Supplement , pp. S19-S22.
http://www.transplantjournal.com/. print.
ISSN: 0041-1337.
DOCUMENT TYPE: General Review
LANGUAGE: English
AB **Dendritic cells** (DC) constitute a complex system [illegible] uniquely well-equipped antigen-presenting cells that initiate and regulate immune responses. Extensive recent studies have improved our understanding of DC development, differentiation, activation, and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and functional capacity. These factors seem to determine the T-cell stimulating potency and type of T cell response-T helper 1, T helper 2, or regulatory T cell-induced by DC [1]. Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas (CD95) ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of gene transcription regulatory proteins (e.g., nuclear factor-kappaB) can impart **tolerogenic** potential to DC (2). Manipulation of DC by control of their maturation and differentiation, or genetic engineering of these cells to express immunosuppressive molecules, offers potential for therapy of allograft rejection and autoimmune disease. In this brief overview, we outline principles and methods for generation of "**tolerogenic**" DC and [illegible] that may [illegible].

L23 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] allograft survival by **dendritic cells** [illegible] genetically engineered [illegible] NF-kappaB [illegible] synthesis in decoys and [illegible] CTLA4-Ig.
AUTHOR(S): [illegible]; Ashton, A.; Lu, Lunsha; Li, Xiaoyan; Chen, Tianyu; Wang, Lianli; Ma, Linlin; Hackstein, Holger; Bromberg, Jonathan S.; Thomson, Angus W.; Fung, John J.; Qian, Shiguang; Lu, Lina
CORPORATE SOURCE: [illegible] Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, 200 Lothrop Street, Biomedical Science Tower, W1544, Pittsburgh, PA, [illegible]: lui@tmx.upmc.edu USA
SOURCE: Journal of Immunology, [illegible], [illegible], [illegible]. [illegible]. http://www.[illegible].print. [illegible].
DOCUMENT TYPE: Article
LANGUAGE: [illegible]
AB [illegible] **dendritic cells** [illegible]

genetically modified using adenoviral (Ad) vectors to express immunosuppressive molecules that promote T cell unresponsiveness. The success of these DC-targeted therapies of allograft rejection has been limited in part by the potential of the adenovirus to promote DC maturation and the inherent ability of the DC to stimulate maturation following in vivo administration. DC maturation occurs via NF-kappaB-dependent mechanisms, which can be blocked by double-stranded "decoy" oligodeoxyribonucleotides (ODNs) containing binding sites for NF-kappaB. Herein, we describe the combined use of NF-kappaB ODNs and rAd vectors encoding CTLA4-Ig (rAd CTLA4-Ig) to generate stably immature murine myeloid DCs that express the potent costimulation blocking agent, human CTLA4-Ig. CTLA4-Ig-transduced DCs exhibit markedly enhanced stability and time to apoptosis in culture. In vivo, administration of rAd CTLA4-Ig NF-kappaB ODN-treated donor DCs [illegible] prolongs MHC-mismatched [illegible] allograft survival, with long-term [illegible] graft survival in 40% of recipients. In vitro studies responsible for the **tolerogenicity**, which may [illegible] alloreactive apoptosis of alloreactive T cells, do not lead to [illegible] intragraft Th cytokine responses. Use of NF-kappaB antisense oligos in conjunction with rAd encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection or autoimmune disease with minimization of systemic immunosuppression.

L23 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:816872 CAPLUS
DOCUMENT NUMBER: 135:355016
TITLE: The use of **tolerogenic dendritic cells** for enhancing **tolerogenicity** in a host and methods for making the same
INVENTOR(S): Robbins, Paul D.; Li, Ling; Ulano Lakeila, Mirk
PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., 84 pp.
[illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001068819	A	[illegible]	WO 2001-US13661	20010427
WO 2001068819	A3	[illegible]		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

[illegible]

PRIORITY APPLN. INFO.: [illegible]

AB [illegible] **tolerogenic** [illegible] **dendritic cells** [illegible] **tolerogenic** [illegible], [illegible] [illegible] **tolerogenicity** [illegible] [illegible] **tolerogenic** [illegible] [illegible] **tolerogenic** [illegible] [illegible] [illegible] [illegible] **tolerogenic** [illegible] [illegible], [illegible] **tolerogenicity** of

the **tolerogenic** [illegible]
tolerogenicity [illegible]
survival [illegible]

L23 [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] mRNA microarray profiles of **tolerogenic dendritic cells**
AUTHOR(S): Rubio-Pena Cortesini, N.; Fiamma, F.; Ho, E.; Marchetti, P.; LeMaoult, J.; Dalla-Favera, R.; Cortesini, R.
CORPORATE SOURCE: Department of Pathology, Columbia University, New York, NY, USA
SOURCE: Human Immunology (2001), 62(10), 1065-1072
CODEN: HUIMDQ; ISSN: 0198-8859
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Dendritic cells** are crucial to the activation as well as suppression of the immune response. Recent reports have illustrated that ADC [illegible] **tolerogenic**, [illegible] **tolerogenic** DC, the mRNA profile of KC-1 **dendritic cells** [illegible] suppressor cells [illegible] This study now provides evidence that immature **dendritic cells** stimulated by T suppressor cells differentiate [illegible] mature **dendritic cells** with a distinct phenotype. The identification of Ts induced pathways of **dendritic cell** differentiation is crit. to the development of new therapeutic strategies.
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:199192 CAPLUS
DOCUMENT NUMBER: 137:41416
TITLE: Prolongation of cardiac allograft survival using **dendritic cells** treated with NF-.kappa.B decoy oligodeoxynucleotides
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: English
AB **Dendritic cells** [illegible] responses for [illegible] [illegible] [illegible] [illegible] induce or suppress immune [illegible] with NF-.kappa.B-[illegible] **tolerogenicity** has been [illegible] with [illegible] NF-.kappa.B-dependent transcription of [illegible] as well as NF-.kappa.B translocation to the nucleus. In this report, we [illegible] double-stranded [illegible] NF-.kappa.B [illegible]

transcription [illegible]. However, exposure of DC to the [illegible] inhibited lipopolysaccharide (LPS)-induced nitric oxide production, a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B [illegible] selectively suppressed the cell-surface expression of costimulatory mols. without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B [illegible] DC induced allogeneic donor-specific hyporesponsiveness in mixed lymphocyte cultures, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B [illegible]-treated bone marrow-derived DC into allogeneic recipients prior to heart transplantation with [illegible] significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways involved in immune activation by DC may [illegible] therapeutic [illegible] in organ transplantation.

L35 ANSWER 1 OF 2 MEDLINE DUPLICATE 1
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] graft survival by **dendritic cells** [illegible] **NF-kappa B** [illegible] and adenoviral vectors [illegible] CTLA4-Ig.
AUTHOR: [illegible]; Lanshu; Liang Xiaoyan; Chen Zongyou; Wang Jianli; Ma Linlin; Hackstein Holger; Robbins Paul D; Thomson Angus W; Fung John J; Qian Shiguang; Lu Lina
CORPORATE SOURCE: Department of Surgery and Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, University of Pittsburgh, Pittsburgh, PA 15213, USA.
CONTRACT NUMBER: AI41011 (NIAID)
SOURCE: JOURNAL OF IMMUNOLOGY, [illegible] Journal code: [illegible]. ISSN: [illegible].
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; [illegible]
LANGUAGE: English
FILE SEGMENT: [illegible]
ENTRY MONTH: [illegible]
ENTRY [illegible]:

AB [illegible] **dendritic cells** [illegible] gene [illegible] adenoviral (Ad) vectors to express immunosuppressive molecules that promote T cell unresponsiveness. The success of these DCs for therapy of allograft rejection has been limited in part by the potential of the adenovirus to promote DC maturation and the inherent ability of the DC to undergo maturation following in vivo administration. DC maturation occurs via NF-kappaB-dependent mechanisms, which can be blocked by double-stranded "decoy" oligodeoxynucleotides (ODNs) containing binding sites for NF-kappaB. Herein, we describe the combined use of NF-kappaB ODNs and rAd vectors encoding CTLA4-Ig (Ad CTLA4-Ig) to generate stably immature murine myeloid DCs that secrete the potent costimulation blocking agent. These Ad CTLA4-Ig-transduced CpG ODN DCs exhibit markedly impaired allostimulatory ability and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig-transduced donor DCs [illegible]; [illegible] before transplant significantly prolongs MHC-mismatched [illegible]; [illegible] before transplant [illegible] graft survival, which [illegible] **tolerogenicity**, [illegible] NF-kappaB antisense oligonucleotides with Ad CTLA4-Ig gene therapy [illegible] costimulation blockade [illegible] therapy of allograft rejection or [illegible] immunosuppression.

L35 ANSWER 2 OF 2 MEDLINE DUPLICATE 2
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible]
TITLE: Manipulation of **dendritic cells** [illegible] disease.
AUTHOR: Lu Lina; Thomson Angus W
CORPORATE SOURCE: Thomas E. Starzl Transplantation Institute, Department of Surgery, University of [illegible], Pittsburgh, [illegible]
CONTRACT NUMBER: [illegible]
SOURCE: [illegible]

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH:
ENTRY DATE:

AB **Dendritic cells** [illegible] immune system [illegible] initiate and regulate immune responses. [illegible] have improved our understanding of DC development, differentiation, activation, and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and [illegible] function. These factors seem to determine the T-cell stimulatory signals and type of T cell response-T helper 1, T helper 2, or T regulatory- induced by DC (1). Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas [CD95] ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of nuclear transcription regulatory proteins (e.g., nuclear factor-kappaB) can impart **tolerogenic** potential to DC [illegible]. Modulation of DC maturation and their maturation and differentiation, to permit maintenance of these cells to express tolerogenic properties, offers potential for therapy of allogeneic [illegible] and autoimmune disease. In this brief overview, we outline [illegible] "**tolerogenic**" DC [illegible] in experimental models. Space [illegible]

I25 [illegible] DUPLICATE 3
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible] PubMed ID: 10943964
TITLE: Prolongation of cardiac allograft survival using **dendritic cells** treated with NF-kB decoy oligodeoxyribonucleotides.
COMMENT: Erratum in: Nat Med [illegible] Sep; [illegible]
Erratum in: Zhang X [corrected to Chen W]
AUTHOR: Giannoukakis N; Phillips B A; Finen S; Chen W; Feng L; Hanaway J; Li W; Thomson A W; Fung J J; Rabinovich P D; Lu L
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, Pennsylvania [illegible], USA.
CONTRACT NUMBER: [illegible]
SOURCE: [illegible]
PUB. [illegible]
[illegible]
LANGUAGE:
FILE SEGMENT:
ENTRY MONTH:
ENTRY DATE:

AB **Dendritic cells** [illegible] immune system [illegible] initiate and regulate immune responses. [illegible] have improved our understanding

tolerogenicity

L25 ANSWER 4 OF 6 MEDLINE
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE: dendritic cells by T(S)
: the critical role of inhibitory receptors ILT3 and
.
COMMENT: Comment in: Nat Immunol. 2002 Mar;3(3):215-7
AUTHOR: ; Gilbertie B; Manavalan J S; Yuan J; Coloval A
; Blanca F; Lederman S; Solomma M; Cortesini R;
-Perera B; -
CORPORATE SOURCE: Department of Pathology, Columbia University, New York, NY 10032, USA.
SOURCE: Nat Immunol, (2002 Mar) 3 (3) 237-43.
Journal code: 100941354. ISSN: 1529-2908.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (CLINICAL TRIAL)
LANGUAGE: English
FILE SEGMENT:
ENTRY MONTH:
ENTRY DATE:

AB
family of
dendritic
cells.
- allogeneic-specific T
ILT3 and ILT4 on
dendritic cells,
tolerogenic. Tolerogenic
show reduced expression of costimulatory molecules and induce
allogeneic hyporesponsiveness in CD4+ T helper cells. Studies of human
circulating DC cells, which include the type-2 blood DC and ILT3 and ILT4
donor APCs. These findings
regulation.

L25 ANSWER 5 OF 6
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE: tolerogenic dendritic
cells tolerogenicity

INVENTOR(S): [illegible] for making the same
Morelli, Adrian E.; Larregina, Lina; Giannoukakis, Nick
PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

W: [illegible]
RW: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 20020048604	A1	20020425	US 2001-944815	20010427

PRIORITY APPLN. INFO.: US 2000-200479P P 20000428

AB The present invention relates to a **tolerogenic** mammalian **dendritic cells** (DCs) and methods for the prodn. of the **tolerogenic** DCs. In addn., the present invention provides a method for enhancing **tolerogenicity** in a host comprising administering the **tolerogenic** mammalian DCs of the present invention to the host. The **tolerogenic** DCs of the present invention comprise an isolated nucleic acid which can be used to inhibit **NF-.kappa.B** activity. The **tolerogenic** [illegible] which does not affect the **tolerogenicity** of the **tolerogenic** DCs when present therein. Enhancing **tolerogenicity** in a host is useful for prolonging the survival of grafts and for treating inflammatory related diseases, such as autoimmune diseases.

L[illegible] ANSWER [illegible] OF [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] mRNA microarray profiles of **tolerogenic dendritic cells**
AUTHOR(S): Suciu-Foca Cortesini, N.; Fiorina, P.; Ho, E.; Ciubotariu, R.; LeMaoult, J.; Dalla-Favera, R.; Cortesini, R.
CORPORATE SOURCE: Department of Pathology, Columbia University, New York, NY, USA
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]

AB **Dendritic cells** [illegible] **tolerogenic**, [illegible] **tolerogenic** [illegible] **dendritic cells** [illegible] dendritic cells [illegible]

dendritic cells

dendritic cell

L15 ANSWER 1 OF 2 [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible]

AUTHOR(S): [illegible]; Beckman, C. Andrew; Qian, Shiguang; [illegible]; Fung, John J.; Robbins, Paul D.; [illegible]

CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA

SOURCE: Molecular Therapy (2000), 1(5, Pt. 1), 430-437
CODEN: MTOHCK; ISSN: 1525-0016

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Dendritic cells (DC) classically promote immune responses but can be manipulated to induce antigen-specific hyporesponsiveness in vitro. The expression of costimulatory mols. (CD40, CD86, CD80) at the DC cell surface correlates with their capacity to induce or suppress immune responses. Expression of these mols. is assocd. with NF-.kappa.B-dependent transcription in DC. **tolerogenicity** has been assocd. with impaired NF-.kappa.B-dependent transcription of costimulatory genes as well as NF-.kappa.B translocation to the nucleus. In this report, we [illegible] phosphorothioate oligodeoxynucleotides for NF-.kappa.B (NF-.kappa.B ODN) are efficiently incorporated by bone marrow-derived DC and specifically inhibit NF-.kappa.B-dependent transcription of a reporter gene. Moreover, exposure of DC to the oligonucleotide decoys inhibited lipopolysaccharide (LPS)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-.kappa.B ODN selectively suppressed the cell-surface expression of costimulatory mols. without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B ODN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcription pathways involved in immune stimulation by DC using ODN decoy approaches may be one means to promote tolerance in clinical transplantation. [illegible]

REFERENCE COUNT: [illegible] THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. [illegible] ARE AVAILABLE IN THE RE FORMAT

L15 ANSWER [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] **tolerogenic** dendritic cells for [illegible] **tolerogenicity** in a host and [illegible] the same

INVENTOR(S): [illegible]; Thomson, Paul D.; [illegible]; Giannokakis, Nick

PATENT ASSIGNEE(S): [illegible] of Pittsburgh on Behalf of the Commonwealth System of Higher Education, USA

SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

[illegible]

WO [illegible]	A2	[illegible]	WO 2001-US13661	20010427	
WO [illegible]	A3	[illegible]			

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US [illegible] A1 [illegible] [illegible] [illegible]

PRIORITY APPLN. INFO.: [illegible]

AB The present [illegible] **tolerogenic** mammalian [illegible] of the **tolerogenic** DCs. In [illegible], the present invention provides a method for [illegible] **tolerogenicity** in a host comprising administering the **tolerogenic** mammalian DCs of the present invention to the host. The **tolerogenic** DCs of the present invention comprise an oligodeoxyribonucleotide (ODN) which has one or more NF-.kappa.B binding sites. The **tolerogenic** DCs of the present invention may further comprise a viral vector, and preferably an adenoviral vector, which does not affect the **tolerogenicity** of the **tolerogenic** DCs when present therein. Enhanced **tolerogenicity** in a host is useful for prolonging foreign graft survival and for treating inflammatory related diseases, such as autoimmune diseases.

L14 ANSWER [illegible] OF 16
ACCESSION NUMBER:
TITLE:
INVENTOR(S):
PATENT ASSIGNEE(S):
SOURCE: [illegible] pp., Cont.-in-part of U.S. Ser. No. 710,802, abandoned.
CODEN: [illegible]
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6469144	B1	20021022	US 1997-[illegible]	[illegible]
CA 2249206	AA	19971009	CA 1997-2249206	[illegible]
US 2002128116	A1	[illegible]	[illegible]	[illegible]
US [illegible]	A1	[illegible]	[illegible]	[illegible]

PRIORITY APPLN. INFO.: [illegible] A [illegible]
[illegible] B [illegible]
[illegible] A1 [illegible]
[illegible] A3 19970331
[illegible] B1 19990430

AB The author discl[illegible] cloning and sequence characterization for the tumor necrosis factor family member Apo-3 and Apo-2 ligand inhibitor (Apo-2LI), an extracellular fragment of Apo-3 generated by alternative splicing. In addn., the author discloses the apoptotic function of Apo-3, a partial characterization of its signaling pathway, and tissue specificity for its expression.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:770147 CAPLUS
DOCUMENT NUMBER: 137:[illegible]
TITLE: Apo-3 protein, a [illegible] member of TNFR family inducing apoptosis and related [illegible] and therapeutics
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible] pp.
CODEN: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US [illegible]	B1	[illegible]	US [illegible]	[illegible]
US 2002165[illegible]	A1	[illegible]	US [illegible]	[illegible]

PRIORITY APPLN. INFO.: US [illegible] B [illegible]
[illegible] B [illegible]
[illegible] B1 [illegible]

AB Novel polypeptides, designated Apo-[illegible], which are capable of inducing apoptosis [illegible]. Apo-[illegible] exhibits sequence [illegible], and certain extracellular [illegible]. The cDNA for Apo-[illegible] is [illegible] and [illegible] Apo-[illegible] [illegible]

CPP32/yama. Apo-1 can activate NF-kappa.B activation.
REFERENCE COUNT: 2[illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 3 OF 10 CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6414233	B1	20020903	US 1999-314691	19990519
PRIORITY APPLN. INFO.:			US 1998-85997P P	19980519
			US 1998-99066P P	19980903

OTHER SOURCE(S): MARPAT 137:116214
AB The invention provides novel saponin mixts. and complex. which are isolated from the species Acacia victoriae and methods for their prepn. These complex. may be utilized in therapeutic formulations, to modulate the activity of NF-kappa B, and to treat diseases. The mixts. and complex. of the invention may be used to treat, e.g., cancer and other diseases characterized by undesirable cell proliferation, e.g., psoriasis, restenosis, etc.
REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER [illegible] CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Inhibition of NF-kappa.B by triterpene comp sitions
INVENTOR(S): Patterson, Grant D.; Berliner, Marsha
PATENT ASSIGNEE(S): Research Triangle Pharmaceuticals, USA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO [illegible]	[illegible]	[illegible]	[illegible]	[illegible]
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.:			[illegible]	[illegible]
			[illegible]	[illegible]

OTHER SOURCE(S):

AB The invention provides methods for treating inflammatory
conditions, such as rheumatoid arthritis, psoriasis, and
NF-κB-dependent diseases. The methods comprise administering
therapeutic agents that, directly or indirectly, inhibit
transcription factor NF-κB. The agents may include
triterpenoids, steroids, lipids, and synthetic and
semisynthetic analogs of triterpenoid and steroid
compounds. The agents may also contain additional chem. functionalities.
Methods of identifying compds. that prevent and treat a wide range of
inflammatory conditions, esp., pro-inflammatory inflammatory conditions are
described.

L14 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:391866 CAPLUS
DOCUMENT NUMBER: 136:396944
TITLE: Preparation of T cell like expressing HIV Tat and
CCR5 and the use of the cell in drug screening
INVENTOR(S): Miyake, Hiroshi; Ikawa, Yuki; Inoue, Hideki
PATENT ASSIGNEE(S): Takara Bio Inc., Japan
SOURCE: U.S. Pat. Appl. Publ.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002040644	A1	20020523	WO 2001-JP10018	20011116

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002014307 A5 20020527 AU 2002-14307 20011116
PRIORITY APPLN. INFO.: JP 2000-350116 A 20001116
JP 2001-[illegible] A 20010[illegible]
WO 2001-JP10018 W 20011116

AB A [illegible] is provided to the expression of HIV
[illegible] and CCR5 [illegible] a drug [illegible]
[illegible] a [illegible] is also provided.

REFERENCE COUNT: [illegible] THERE ARE [illegible] RE FORMATS AVAILABLE FOR THIS RECORD. ALL [illegible] AVAILABLE IN THE RE FORMAT

L14 ANSWER [illegible] CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] Adrenal survival using
[illegible] with NF-κB
[illegible]
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]

AB Dendritic cells (DC) classically prime for immune responses but can be manipulated to induce antigen-specific hyporesponsiveness in vitro. The expression of costimulatory mols. [illegible] on the DC cell surface correlates with their capacity to induce or suppress immune responses. Expression of these mols. is controlled, in part, by NF-.kappa.B-dependent transcription of their genes. DC tolerogenicity has been assocd. with impaired NF-.kappa.B-dependent transcription of costimulatory genes as well as NF-.kappa.B [illegible] In this study, [illegible] NF-.kappa.B [illegible] bone marrow-derived DC [illegible] Moreover, exposure [illegible] lipopolysaccharide (LPS)-induced [illegible] DC maturation. Treatment of bone marrow-derived DC precursors with NF-.kappa.B ODN selectively suppressed the LPS-induced expression of costimulatory mols. without interfering with MHC class I or class II expression. Furthermore, NF-.kappa.B ODN [illegible] antigen-specific anergy in naive T cells, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways involved in immune stimulation in DC using ODN decoy approaches could be one means to promote tolerance induction in organ transplantation. (c) 2008 Academic Press.

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER [illegible]

ACCESSION NUMBER: [illegible]

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] and methods for making the same

INVENTOR(S): Robbins, Paul D.; Lu, Lina; Giannoukakis, Nick

PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA

SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001083713	A2	[illegible]	[illegible]	[illegible]
WO 2001083713	A3	[illegible]		

DESIGNATED STATES: [illegible]

PRIORITY APPLN. INFO.: [illegible]

AB [illegible]

NF-.kappa.B activity. In another aspect of the present invention, a recombinant viral vector, preferably an adenoviral vector, which expresses a GSK-3 inhibitory peptide is provided. Administration of the viral vector to a host is useful for preventing or treating inflammatory related diseases, such as arthritic diseases.

L14 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:496442 CAPLUS
DOCUMENT NUMBER: 135:67191
TITLE: Inhibition of glycogen synthase kinase 3 beta. (GSK-3.beta.) for inhibiting NF-.kappa.B, and therapeutic use
INVENTOR(S): Hoeflich, Klaus; Lu, Yuan; Woodgett, Jim
PATENT ASSIGNEE(S): The Ontario Cancer Institute, Can.
SOURCE: PCT Int. Appl., 71 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001047552	A1	20010705	WO 2000-CA1578	20001221
W: AE, AG, AL, AM,				
RW: AT, BE, CH, CY, DE, DK, EA, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
EP 1244461	A2	20021002	EP 2000-986051	20001221
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
US 2001053351	A1	20011220	US 2000-747652	20001221
PRIORITY APPLN. INFO.:			US 1999-173064P	P 19991224
			WO 2000-CA1578	W 20001221

AB The activity of NF-kB is mediated through the activity of IKK-.alpha. and IKK-.beta. activity. Inhibition or down-regulation of IKK-.beta. results in decreased NF-kB activity. Inhibitors of IKK-.beta. and NF-kB may be used [illegible] inflammatory and proliferative diseases. [illegible] GSK-3 inhibitors, where administration is in conjunction with [illegible] TNF [illegible]. [illegible] of IKK-.beta. activation are also provided.

L14 ANSWER [illegible] CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Regulation of HIV-1 transcription
AUTHOR(S): Boswell, Kenneth A.; Rafferidin, Mohammed
CORPORATE SOURCE: Department of Immunology, Microbiology, Rush [illegible], Chicago, IL, [illegible], USA
SOURCE: [illegible]
CODEN: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB [illegible]

learned about the transcriptional regulation of the HIV-1 genome in infected cells. It has been demonstrated that HIV-1 transcription depends on a varied and complex interaction of host cell transcription factors with the viral long terminal repeat (LTR) promoter. The regulatory elements within the LTR interact with constitutive and inducible transcription factors to direct the assembly of a stable transcription complex that stimulates multiple rounds of transcription by RNA polymerase II (RNAPII). However, the majority of these transcription factors [illegible] promoter, which is [illegible] stem-loop (TAR) structure at the 5' end of all viral transcripts. The Tat-TAR interaction is critical for efficient [illegible] In this review we describe what is known about the [illegible] HIV-1 LTR. This review summarizes our current knowledge and understanding of the regulation of HIV-1 transcription in [illegible] and highlights the important contributions human lentivirus gene regulation has made to our general understanding of the transcription process.

REFERENCE COUNT: 107 THERE ARE 107 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:657226 CAPLUS

DOCUMENT NUMBER: 132:161869

TITLE: Long terminal repeat promoter/enhancer activity of different subtypes of HIV type 1

AUTHOR(S): Naghavi, Mojgan H.; Schwartz, Stefan; Sonnerborg, Anders; Vahlne, Anders

CORPORATE SOURCE: [illegible]

SOURCE: [illegible]

CODEN: AMRRA7; ISSN: 0889-2229

PUBLISHER: Mary Ann Liebert, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Transcription of the HIV-1 provirus genome is regulated by a complex interplay between viral regulatory proteins and cellular transcription factors that interact with the viral long terminal repeat (LTR) region of HIV-1. However, several cellular transcription factors have been identified that can interact with the HIV-1 LTR; the significance of all of these factors is not clearly understood. In this study the authors have characterized the LTR region of different subtypes of HIV-1 with regard to nucleotide sequence and promoter activity. The LTR [illegible] HIV-1 from peripheral [illegible] originating from [illegible]

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER [illegible] ON STN
ACCESSION NUMBER:
DOCUMENT NUMBER:
TITLE: [illegible]-.kappa.B p50 subunit binding to DNA duplexes, [illegible] pyrophosphate
AUTHOR(S): [illegible]
CORPORATE SOURCE: A.N. Belozersky Institute of Physical and Chemical Biology and Department of Chemistry, Moscow State University, Moscow, 119899, Russia
SOURCE: FEBS Letters (1996), 381(1,2), 35-8
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A new express technique based on the use of biotinylated DNA duplexes containing non-nucleotide linkers of variable length between the [illegible] was developed. The method was applied to study the binding of NF-.kappa.B p50 subunit to DNA duplexes containing the [illegible] of the palindromic sequence in HIV-1 5'-[illegible]-3'. Using oligonucleotide duplexes with the [illegible] subunit complex with the oligonucleotide sequence. The crosslinking of NF-.kappa.B p50 subunit to the DNA duplex only, a monosubstituted pyrophosphate [illegible] was successfully performed.

L14 ANSWER [illegible] ON STN
ACCESSION NUMBER: 1994:690026 CAPLUS
DOCUMENT NUMBER: 121:296026
TITLE: Determination of the binding of transcription factors to nucleic acids by immunoassay
INVENTOR(S): Doppler, Clemens; Hilpert, Matthias; Stockinger, Hubertus
PATENT ASSIGNEE(S): Boehringer Mannheim G.m.b.H., Germany
SOURCE: Eur. Pat. Appl., 9 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	19940414
[illegible]				
[illegible]				
[illegible]	[illegible]	[illegible]	[illegible]	19930416

PRIORITY APPLN. INFO.: [illegible] 19930416
AB [illegible] binding of transcription factors to nucleic acids is detd. by (a) [illegible] a biotinylated nucleic acid binding to the transcription factor in a solid phase; (b) incubating the transcription factor with the nucleic acid; (c) adding a labeled antibody directed against the nucleic acid or the transcription factor under conditions which [illegible]; (d) sepg. the solid and liq. phases; and (e) measuring the amt. of label in either phase. Thus, binding of transcription factor NFKB in nuclear ext. of PMA-PHA-activated Jurkat cells to a synthetic oligonucleotide contg. a binding site for NFKB was detd. by [illegible] the oligonucleotide to streptavidin-coated microtiter plates, incubating with NFKB, adding monoclonal antibody against NFKB, and measuring the [illegible].

L14 ANSWER [illegible] ON STN

ACCESSION NUMBER: 1994:[illegible]
DOCUMENT NUMBER: [illegible]
TITLE: In vitro study [illegible] Sp1, NF-.kappa.B/Rel, and AP1 in phorbol 12-myristate 13-acetate-mediated HIV-1 long terminal repeat activation
AUTHOR(S): Li, Yuchi; Mak, [illegible]; Franza, B. Robert, Jr.
CORPORATE SOURCE: Cold Spring Harbor Lab., Cold Spring Harbor, New York, NY, [illegible], USA
SOURCE: [illegible]
[illegible]
PUBLISHER: [illegible] Biochemistry and Molecular [illegible]
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We examd. the cooperative activity between the Sp1 and NF-.kappa.B/Rel sites of the human immunodeficiency virus type 1 long terminal repeat in response to phorbol 12-myristate 13-acetate (PMA) stimulation in an in vitro transcription assay. Sp1 sites alone do not account for the activation induced by PMA. When mutations in Sp1 sites were combined with mutations in the NF-.kappa.B/Rel sites, a dramatic redn. in PMA-induced transcriptional activity was obsd. This redn. was much greater than the redn. assocd. with mutations involving only the NF-.kappa.B/Rel sites. This finding suggests that there is functional cooperation between Sp1 and NF-.kappa.B/Rel and that this is one possible mechanism for transcription activation by NF-.kappa.B/Rel. The three AP1 sites in the [illegible] regulatory region of the long terminal repeat, however, seem to be uninvolved in the earliest moments of transcriptional activation by PMA.

L14 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Transcription factor AP-4 regulates human immunodeficiency virus type 1 gene expression
AUTHOR(S): [illegible], [illegible]; [illegible], Adam, P.; Duckett, Colin [illegible]; [illegible], Gary [illegible]
CORPORATE SOURCE: Howard Hughes Medical Institute, University Michigan, Ann Arbor, MI, 48109-0650, USA
SOURCE: Journal of Virology (1994), 68(10), 6820-3
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Human immunodeficiency virus type 1 (HIV-1) gene expression is regulated by an enhancer region composed of multiple potential cis-acting regulatory sites. Here, we [illegible] in the HIV-1 long terminal [illegible]. One site is [illegible] elements, and a [illegible] footprinting [illegible]

L14 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] zinc finger protein [illegible] promoter and [illegible] major histocompatibility complex class I enhancer
AUTHOR(S): [illegible]

CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB The [illegible] DNA [illegible] DNA-binding proteins (AT-BP1 and AT-BP2) were [illegible]. In particular, these proteins, which bind to the B-domain of the .alpha.1-antitrypsin promoter, an element which is important for the liver-specific expression of .alpha.1-antitrypsin. Anal. of the cDNA sequences encoding these proteins reveals that both contain 2 zinc fingers of the Cys2-His2 type followed by a highly acidic stretch of 20 amino acids. AT-BP1 contains a 2nd putative DNA-binding domain consisting of an 8-fold repeat of a SPKK (Ser-Pro-Lys/Arg-Lys/Arg) motif. Both proteins bind to the NF.kappa.B recognition site in the HIV gene enhancer with significant higher affinity than to the .kappa. immunoglobulin gene enhancer, or to the B-domain of the .alpha.1-antitrypsin gene promoter. Anal. of mRNA expression shows that AT-BP1 and AT-BP2 are expressed in all the tissues exami. While the physiol. roles of AT-BP1 and AT-BP2 remain to be elucidated, their predicted [illegible] structure [illegible] DNA-binding specificities suggest [illegible].

L14 AN [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: UV-induced DNA damage is an intermediate step in UV-induced expression of human immunodeficiency virus type 1, collagenase, c-fos, and metallothionein
AUTHOR(S): Stein, Bernd; Rahmsdorf, Hans Jobst; Steffen, Anja; Litfin, Margarethe; Herrlich, Peter
CORPORATE SOURCE: Inst. Genet. Toxikol., Kernforsch. Karlsruhe, Karlsruhe, D-7500/1, Fed. Rep. Ger.
SOURCE: Molecular and Cellular Biology (1989), 9(11), 5169-81
CODEN: MCEBD4; ISSN: 0270-7306
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The primary target of relevant UV absorption, the pathways leading to gene activation, and the elements involved in the UV-induced signal in the human immunodeficiency virus type 1 (HIV-1) long terminal repeat, in the gene coding for [illegible], and in the [illegible] were investigated. [illegible] UV-induced [illegible] DNA damage [illegible] HIV-1, between positions -72 and -61 of the [illegible] site, and between positions -320 and -299 of fos). These [illegible] appear to [illegible] and bind different [illegible]; a member of the NF.kappa.B family binds to the HIV-1 enhancer, the factor Sp1 and Ets (AP-1) binds to the [illegible] enhancer, and the [illegible] and pu2 bind to fos. UV-induced activities of the factors recognizing the HIV-1 and collagenase enhancers are augmented in extracts from UV-treated cells. The increase in activity is due to post-translational modification. Whereas AP-1 resides in the nucleus and must be modified there, NF.kappa.B is activated in the cytoplasm, indicating the existence of a cytoplasmic signal transduction pathway triggered by UV-induced DNA damage. In addition to activation, new synthesis of AP-1 is induced by UV irradiation.

PALM INTRANET

Day : Thursday
Date: 11/21/2002
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Additionally, enter the **first few letters** of the Inventor's First name.

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PALM INTRANET

Day : Thursday
Date: 11/21/2002
Time: 10:39:03

Inventor Name Search

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Lu	Lina	Search

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Back to PALM ASSIGNMENT OASIS Home page

L6 ANSWER 1 OF 9 BIOSIS COPYRIGHT 2[illegible] BIOLOGICAL ABSTRACTS INFORMATION [illegible]
ACCESSION NUMBER: 2002:345247 BIOSIS
DOCUMENT NUMBER: PREV[illegible]
TITLE: [illegible]
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: Article
LANGUAGE: English
AB Background: The [illegible] associated with the general immune suppression resulting from current clinical immunosuppressive therapies continue to plague transplant recipients as well as jeopardize allograft survival. Methods: The present study utilized allochimeric class I MHC antigens (alphalh u70-77-RT1.Aa) bearing only four donor RT1.Au polymorphic amino acids (a.a.; His[70], Val[73], Asn[74], and Asn[77]) superimposed on the recipient RT1.Aa background to induce transplantation tolerance in the rat cardiac transplant model. Results: Oral delivery of alphalh u70-77-RT1.Aa protein alone (days 1-[illegible]) induced tolerance, as evidenced by inhibition of both acute and chronic rejection processes. Delivery of alphalh [illegible]-RT1.Aa with therapeutic doses of cyclosporine (CsA) also prevented [illegible], tolerance readily developed after treatment with [illegible]. [illegible] ELISA-based assays showed that [illegible] elevated [illegible] and elevated [illegible]. Adoptive transfer experiments [illegible] T cells mediated tolerance. The same T cells displayed diminished T cell receptor (TCR)-driven signaling via extracellular regulated kinase, AP-1, and NF-kappaB, as well as the common gamma-chain (gammac) cytokine-receptor-induced signaling by Janus kinase 3 (Jak3)/stimulators and activators of transcription Stat/5 pathways. Tolerance induction was prevented in vivo by inhibition of signal 2 by CTLA4Ig or of signal 3 by either rapamycin, which disrupts the mammalian target of rapamycin, or AG490, which inhibits Jak3. Finally, partial or complete tyrosine phosphorylation of Tyk2 was [illegible] in alloantigen-specific T cell clones in response to **tolerogenic** versus immunogenic peptides, respectively. Conclusions: [illegible] induction by allochimeric proteins is achieved by partial TCR activation in the presence of signals 2 and 3, resulting in a skewed Th2 phenotype.

L6 ANSWER 2 OF 9 [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible]
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: Editorial Review
LANGUAGE: English
AB Dendritic cells (DC) constitute a unique system of [illegible] antigen-presenting [illegible]

[illegible] understanding of DC development, [illegible], and function. DC exist as distinct [illegible] that differ in their lineage affiliation, surface molecule expression, and [illegible] function. These factors seem to determine the T-cell polarizing signals and type of T cell response-T helper 1, T helper 2, or T regulatory-induced by DC (1). Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas (CD95) ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of [illegible] transcription regulatory proteins [illegible] **tolerogenic** [illegible] their [illegible] cells to [illegible], offers potential for therapy of [illegible]. In this brief overview, we outline principles and methods for generation of **"tolerogenic"** DC and outcomes that have been reported in experimental models. Space constraints limit literature citations.

L6 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
ACCESSION NUMBER: [illegible] BIOSIS
DOCUMENT NUMBER: PREV200300167608
TITLE: Reduced expression of NF-AT and NF-kappaB transcription factors in tolerant recipients treated with **tolerogenic** allochimeric donor/recipient class I MHC protein.
AUTHOR(S): Akioka, K. (1); Kirkin, R.; Wang, M.; Tian, L.; Yu, J.; Stepkowski, S. M.; Kahan, B. D.
CORPORATE SOURCE: (1) Division of Immunology and Organ Transplantation, University of Texas Medical School at Houston, [illegible] St, Houston, TX, [illegible]
SOURCE: Transplantation Proceedings, [illegible]
[illegible]
[illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
SUMMARY LANGUAGE: [illegible]

L6 ANSWER 4 OF 4 BIOSIS COPYRIGHT [illegible] BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: [illegible] BIOSIS
DOCUMENT NUMBER: PREV[illegible]
TITLE: Marked prolongation of cardiac allograft survival by dendritic cells genetically engineered with NF-kappaB oligodeoxyribonucleotide decoys and adenoviral vectors encoding CTLA4-Ig.
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]

DOCUMENT TYPE: Article
LANGUAGE: English
AB Bone marrow-derived dendritic cells (DCs) can be genetically engineered using adenoviral (Ad) vectors to express immunosuppressive molecules that promote T cell unresponsiveness. The promise of these DCs for therapy of allograft rejection has been limited in part by the toxicity of the adenovirus vector to DCs and by the inherent ability of the DCs to [illegible] Here we describe a method for the generation of [illegible] DCs by [illegible] blocking agent. Here, we describe the combined use of NF-kappaB decoy ODN and Ad encoding CTLA4-Ig (Ad CTLA4-Ig) to generate stably immunosuppressive DCs that secrete the potent costimulation blocking agent. These Ad CTLA4-Ig-transduced ODN DCs exhibit markedly impaired allostimulatory ability and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL/10; B10/H-2k) before transplant significantly prolongs MHC-mismatched (BALB/c; BALB/H-2d) vascularized heart allograft survival, with long-term (>100 days) donor-specific graft survival in 40% of recipients. The mechanism(s) responsible for DC **tolerogenicity**, which may involve activation-induced apoptosis of alloreactive T cells, do not lead to skewing of intragraft Th cytokine responses. Use of NF-kappaB antisense decoys in conjunction with rAd encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection or autoimmune disease with minimization of systemic immunosuppression.

L6 ANSWER [illegible] OF [illegible] CAPLUS COPYRIGHT [illegible] ACS
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **tolerogenic** [illegible]
[illegible] **tolerogenicity** [illegible]
[illegible]
INVENTOR(S): [illegible]
PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001068718	A2	[illegible]	[illegible]	[illegible]
WO 2001068718	A3	[illegible]		

W: [illegible]

RW: [illegible]

PRIORITY APPLN. INFO.: [illegible]

AB The present invention relates to **tolerogenic** [illegible] **tolerogenic** DCs. In addition, the present invention provides a method for enhancing **tolerogenicity** [illegible] administering the **tolerogenic** [illegible] invention to the host. The **tolerogenic** [illegible] invention comprises [illegible]

NF-.kappa.B [illegible]. The **tolerogenic** [illegible] the present invention may [illegible], and preferably an [illegible] the **tolerogenicity** of the **tolerogenic** [illegible] present [illegible]. Enhanced **tolerogenicity** [illegible] useful for prolonging foreign graft survival and [illegible] inflammatory related diseases, such as auto-immune diseases.

L6 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2[illegible] ACS

ACCESSION NUMBER: 2001:922841 CAPLUS

DOCUMENT NUMBER: 137:45987

TITLE: The role of Stat5 in the induction of [illegible] T cells in transplantation tolerance

AUTHOR(S): Stepkowski, S. M.; Kirken, R. A.; [illegible]; Trawick, B. W.; [illegible]; [illegible]; [illegible]; Tian, L.; [illegible]; [illegible]

CORPORATE SOURCE: [illegible] Department of [illegible], University of Texas, Houston, TX, USA

SOURCE: [illegible]

PUBLISHER: [illegible]

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The roles of extracellular-regulated kinase 2 (Erk2), NF-.kappa.B, AP-1, Janus tyrosine kinase 3 (Jak3), and stimulators and activators of transcription 5 (Stat5) in mediating transplantation tolerance were studied. Tolerant recipients that had carried functional Wistar-Furth (WF) grafts for more than 100 days were rechallenged with a second WF heart. Purified T cells from spleens and lymph nodes of rejectors and tolerant T cells showed a significant increase in Jak3 expression. The presence of phosphorylated Erk2 and the expression of Jak3 indicated that the tolerant animals have actively reacting alloantigen-specific T cells, but that their response was distinct from that in nonactivated T cells of syngeneic grafts or fully activated T cells in rejectors. Tolerant T cells showed almost undetectable AP-1, NF-.kappa.B, and Stat5 DNA binding activities. The [illegible]-2 clone stimulated with [illegible] peptide showed potent Stat5 DNA binding [illegible] IL-2. On the other hand, the [illegible] **tolerogenic** [illegible] peptide [illegible] Stat5 [illegible] partial TCR [illegible].

REFERENCE COUNT: [illegible] THERE ARE [illegible] CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER [illegible] OF [illegible] CAPLUS COPYRIGHT [illegible] ACS

ACCESSION NUMBER: [illegible] CAPLUS

DOCUMENT NUMBER: [illegible]

TITLE: [illegible] mRNA microarray profiles of **tolerogenic** [illegible] cells

AUTHOR(S): [illegible]-Foca Cortesini, N.; [illegible], F.; [illegible]; Ciubotariu, R.; [illegible]; [illegible]; Cortesini, R.

CORPORATE SOURCE: Department of [illegible], Columbia University, New York, NY, USA

SOURCE: [illegible]

PUBLISHER: [illegible]

DOCUMENT TYPE: [illegible]

LANGUAGE: [illegible]

AB [illegible] **tolerogenic**, [illegible]

changes occurring in **tolerogenic** AP[illegible], the mRNA profiles of NF-[illegible] dendritic cells exposed to allogeneic T helper and T suppressor [illegible] were analyzed. This study now provides evidence that immature dendritic cells stimulated by T suppressor cells attain a mature dendritic cell with a distinct phenotype. The [illegible] of NF-[illegible] pathways of dendritic cell differentiation in vivo. The development of new therapeutic strategies

REFERENCE COUNT: [illegible] THERE ARE [illegible] REFERENCES AVAILABLE FOR THIS RECORD. ALL REFERENCES ARE AVAILABLE IN THE RE FORMAT

L6 ANSWER [illegible] OF [illegible] MEDLINE
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Prolongation of cardiac allograft survival using dendritic cells treated with NF-.kappa.B decoy oligodeoxyribonucleotides
AUTHOR(S): Hackstein, Nils; Denhan, C. Andrew; Qian, Shiguang; Zhou, Zhongyou; Peng, Lansha; Harnaha, Jo; Li, Wei; Thomson, Angus W.; Fung, John J.; Robbins, Paul D.; Lu, Lina
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA
SOURCE: Molecular Therapy [1999], 1, Pt. 1, 41-48
CODEN: MTOHCK; ISSN: 1525-0016
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal

L8 ANSWER [illegible] [illegible] ON STN ABSTRACTS INCLUDE
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **dendritic cells** [illegible]
[illegible] induction in transplantation and autoimmune disorders.
AUTHOR(S): [illegible]
CORPORATE SOURCE: [illegible] Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, 200 Lothrop Street, E1654, Biomedical Science Tower, Pittsburgh, PA, 15213: Lul@msx.upmc.edu: USA
SOURCE: Transplantation (Baltimore), (January 15, 2002) Vol. 73, No. 1 Supplement , pp. S16-S18.
http://www.transplantjournal.com/. print.
ISSN: [illegible].
DOCUMENT TYPE: General Review
LANGUAGE: [illegible]
AB **Dendritic cells** [illegible]
[illegible] have improved our understanding of [illegible], [illegible], and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and biological function. These factors seem to determine the T-cell polarizing signals and type of T cell response-T helper 1, T helper 2, or T regulatory-induced by DC (1). Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas (CD95) ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines , and inhibition of gene transcription regulatory proteins (e.g., nuclear factor-kappaB) can impart **tolerogenic** potential. [illegible] Manipulation of DC by control of their maturation and differentiation, [illegible] cells to express [illegible], therapy [illegible] therapy of [illegible]. [illegible] "**tolerogenic**" [illegible]

L8 ANSWER [illegible] [illegible] ON STN ACCESSION NUMBER: [illegible] DOCUMENT NUMBER: [illegible] TITLE: [illegible] **dendritic cells** [illegible] AUTHOR(S): [illegible] CORPORATE SOURCE: [illegible] SOURCE: [illegible] PUBLISHER: [illegible] DOCUMENT TYPE: [illegible] LANGUAGE: [illegible] AB [illegible] **dendritic cells** [illegible]

genetically [illegible] immunosuppressive [illegible] stress [illegible] antisense [illegible] antigen-independent mechanisms, [illegible] Ad CTLA4-Ig [illegible] potent costimulation blocking agent. These Ad CTLA4-Ig-transduced ODN DCs exhibit markedly impaired allostimulatory ability and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL10; B10(H-2b)) before transplant significantly prolongs MHC-mismatched (C3HHeJ; C3H(H-2k)) vascularized heart allograft survival, with long-term (>100 days) donor-specific graft survival in 4[illegible] recipients. The mechanism(s) responsible for DC **tolerogenicity**, which may involve activation-induced apoptosis of alloreactive T cells, [illegible] not lead to skewing of intragraft Th cytokine responses. [illegible] antisense decoys in conjunction with mAb encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection [illegible] autoimmune disease with minimal risk of systemic immunosuppression.

L8 ANSWER [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] of **tolerogenic dendritic cells** [illegible] enhancing **tolerogenicity** [illegible] and methods for making the same
INVENTOR(S): [illegible]; Lu, Lina; Giannoukakis, Nick
PATENT ASSIGNEE(S): University of Pittsburgh of the Commonwealth System of Higher Education, USA
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO [illegible]	A	[illegible]	[illegible]	[illegible]
WO [illegible]	A3	[illegible]		

W: [illegible]
RW: [illegible]

PRIORITY APPLN. INFO.: [illegible]

AB The present invention relates to a **tolerogenic** mammalian **dendritic cells** [illegible] and methods for the preparation of **tolerogenic** DCs. In addn., the present invention provides a method for enhancing **tolerogenicity** in a subject by administering the **tolerogenic** [illegible] of the present invention to the host. The **tolerogenic** [illegible] of the present invention comprises an isolated mammalian [illegible] NF-κB p65 inhibitor. The **tolerogenic** [illegible] of the present invention may be administered, [illegible] and the **tolerogenicity**

the **tolerogenic** DCs when present [illegible]. Manipulation of **tolerogenicity** in a host is useful [illegible] survival and for treating inflammatory [illegible] autoimmune diseases.

L2 ANSWER 4 OF [illegible]
ACCESSION NUMBER: [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: [illegible] **tolerogenic dendritic cells**
AUTHOR(S): [illegible]
CORPORATE SOURCE: Department of Pathology, Columbia University, New York, NY, USA
SOURCE: Human Immunology (2001), 62(10), 1065-1072
CODEN: HUIMDV; ISSN: 0198-8859
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Dendritic cells** are crucial to the activation as well as suppression of the immune response. Previous reports have illustrated that APC interacting with antigen-specific T suppressor cells become **tolerogenic**, inducing T helper anergy. To characterize the molecular changes occurring in **tolerogenic** APC, the mRNA profile of MHC-I **dendritic cells** exposed to allospecific T helper and T suppressor [illegible] analysis. This analysis revealed that immature **dendritic cells** [illegible] **dendritic cells** [illegible] **dendritic cell** [illegible] development [illegible] therapeutic strategies.
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 5 OF [illegible] COPYRIGHT 2001 ACS
ACCESSION NUMBER: [illegible]:199140 CHEMCS
DOCUMENT NUMBER: [illegible]:41141
TITLE: Prolongation of cardiac allograft survival using **dendritic cells** treated with NF-.kappa.B decoy oligodeoxyribonucleotides
AUTHOR(S): Giannoukakis, Nick; Bonham, C. Andrew; Qian, Shiguang; Zhou, Zhongyou; Penn, Lansha; Harmaha, J.; Li, Wei; Thomson, Angus W.; Poli, [illegible]; Robbins, Paul D.; Ma, Libo
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
PUBLISHER: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: [illegible]
AB **Dendritic cells** [illegible] **tolerogenicity** [illegible] dependent transcription [illegible] translocation to the nucleus. In this report, we [illegible] double-stranded [illegible] NF-.kappa.B (NF-.kappa.B [illegible] marrow-derived [illegible]

transcription factor. However, the role of DC in the [illegible] lipopolysaccharide (LPS)-induced nitric oxide [illegible] Treatment of bone marrow-derived DC with NF-.kappa.B [illegible] suppressed the [illegible] expression. Furthermore, NF-.kappa.B ODN DC induced [illegible] in a T-specific hyporesponsiveness in mixed leukocyte cultures, and this was assocd. with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-.kappa.B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-.kappa.B and other transcriptional pathways [illegible] immune [illegible] decoy approaches could be [illegible] organ transplantation.

REFERENCE COUNT: [illegible] THERE ARE [illegible] RECORDS THAT MATCHED YOUR QUERY.